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Preliminary Air Pollution Survey of Chlorine Gas
A Literature Review

Litton Systems, Inc.
Bethesda, MD

Prepared for

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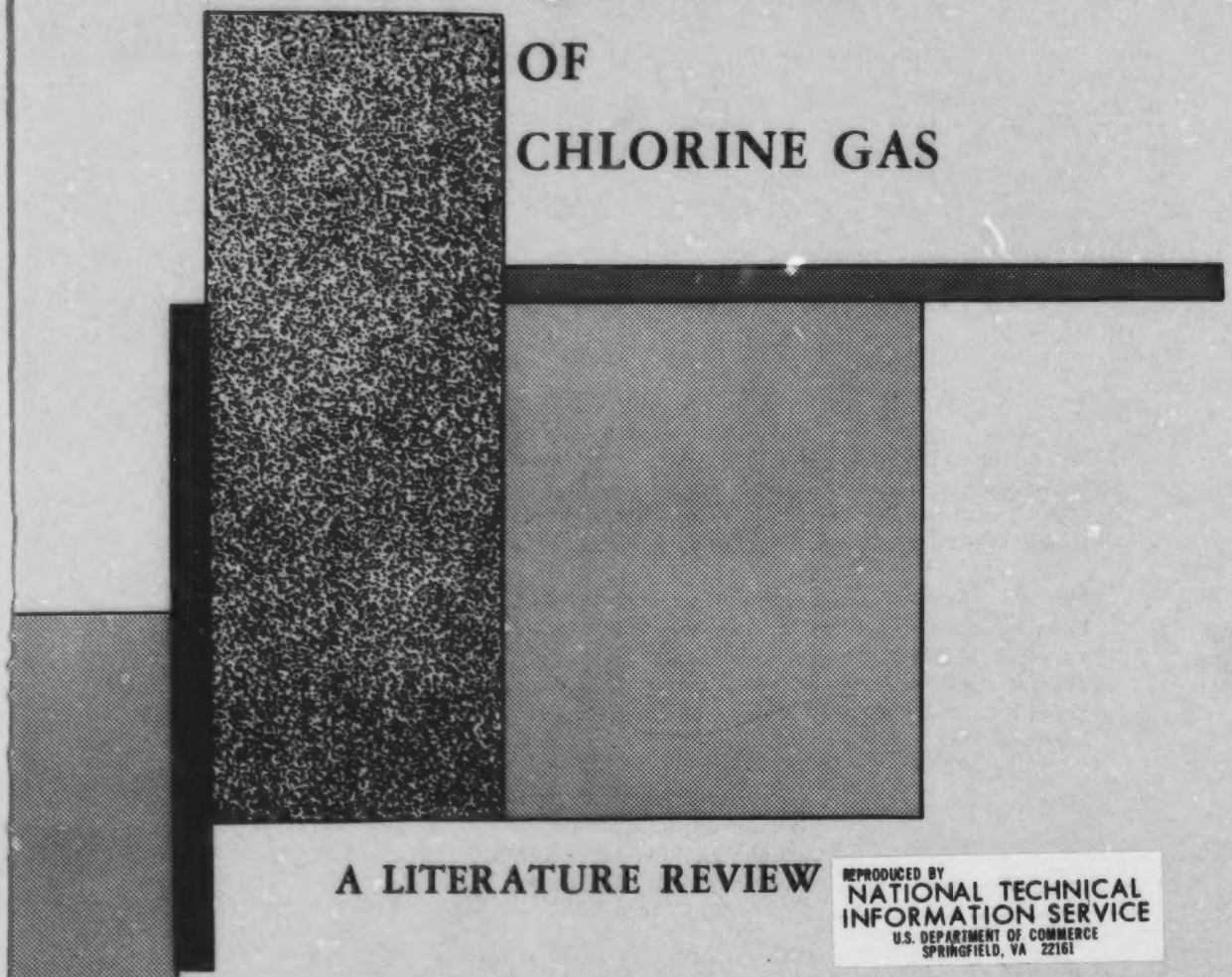

A LITERATURE REVIEW

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PREFACE

This document represents a preliminary literature review which is being used as a basis for further evaluation, both internally by the National Air Pollution Control Administration (NAPCA) and by contractors. This document further delineates present knowledge of the subject pollutant, excluding any specific conclusions based on this knowledge.

This series of reports was made available through a NAPCA contractual agreement with Litton Industries. Preliminary surveys include all material reported by Litton Industries as a result of the subject literature review. Except for section 7 (Summary and Conclusions), which is undergoing further evaluation, the survey contains all information as reported by Litton Industries. The complete survey, including section 7 (Summary and Conclusions) is available from:

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Describes the sources, distribution, effects and proposed solutions for problems relating to the presence of chlorine gas in the atmosphere. Summary type information is presented on the product sources, producers and users of chlorine gas in the U.S., and harmful effects on human, plant and animal life. Brief discussions of possibilities for control of these detrimental effects are included, as well as sections on the economic aspects and methods of analysis. Also present are a lengthy bibliography and an appendix containing tables of related data.

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PRELIMINARY AIR POLLUTION SURVEY OF CHLORINE GAS

A LITERATURE REVIEW

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Litton Systems, Incorporated
Environmental Systems Division

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U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Consumer Protection and Environmental Health Service
National Air Pollution Control Administration
Raleigh, North Carolina
October 1969

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FOREWORD

As the concern for air quality grows, so does the concern over the less ubiquitous but potentially harmful contaminants that are in our atmosphere. Thirty such pollutants have been identified, and available information has been summarized in a series of reports describing their sources, distribution, effects, and control technology for their abatement.

A total of 27 reports have been prepared covering the 30 pollutants. These reports were developed under contract for the National Air Pollution Control Administration (NAPCA) by Litton Systems, Inc. The complete listing is as follows:

Aeroallergens (pollens)
Aldehydes (includes acrolein and formaldehyde)
Ammonia
Arsenic and Its Compounds
Asbestos
Barium and Its Compounds
Beryllium and Its Compounds
Biological Aerosols (microorganisms)
Boron and Its Compounds
Cadmium and Its Compounds
Chlorine Gas
Chromium and Its Compounds (includes chromic acid)
Ethylene
Hydrochloric Acid
Hydrogen Sulfide
Iron and Its Compounds
Manganese and Its Compounds
Mercury and Its Compounds
Nickel and Its Compounds
Odorous Compounds
Organic Carcinogens
Pesticides
Phosphorus and Its Compounds
Radioactive Substances
Selenium and Its Compounds
Vanadium and Its Compounds
Zinc and Its Compounds

These reports represent current state-of-the-art literature reviews supplemented by discussions with selected knowledgeable individuals both within and outside the Federal Government. They do not however presume to be a synthesis of available information but rather a summary without an attempt to interpret or reconcile conflicting data. The reports are

necessarily limited in their discussion of health effects for some pollutants to descriptions of occupational health exposures and animal laboratory studies since only a few epidemiologic studies were available.

Initially these reports were generally intended as internal documents within NAPCA to provide a basis for sound decision-making on program guidance for future research activities and to allow ranking of future activities relating to the development of criteria and control technology documents. However, it is apparent that these reports may also be of significant value to many others in air pollution control, such as State or local air pollution control officials, as a library of information on which to base informed decisions on pollutants to be controlled in their geographic areas. Additionally, these reports may stimulate scientific investigators to pursue research in needed areas. They also provide for the interested citizen readily available information about a given pollutant. Therefore, they are being given wide distribution with the assumption that they will be used with full knowledge of their value and limitations.

This series of reports was compiled and prepared by the Litton personnel listed below:

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Appreciation is expressed to the many individuals both outside and within NAPCA who provided information and reviewed draft copies of these reports. Appreciation is also expressed to the NAPCA Office of Technical Information and Publications for their support in providing a significant portion of the technical literature.

ABSTRACT

Low concentrations of chlorine gas in the atmosphere, e.g. 3,000 $\mu g/m^3$ (1 ppm), can cause irritation of the eyes, nose, and throat; larger doses can cause damage to the lungs and produce pulmonary edema, pneumonitis, emphysema, or bronchitis. Chlorine gas is known to have caused injury and death to humans and animals as well as to have damaged plant life. Its highly corrosive nature suggests the possibility of material damage; however, no instances of damage by atmospheric chlorine have been reported in the literature.

Possible sources of chlorine in the atmosphere are industrial liquefication processing, other industrial uses of chlorine, and accidental leakage during storage or transportation. Production of chlorine has doubled in the past 10 years and is expected to continue at this rate of increase for several years. No information is currently available on the concentrations of chlorine gas in ambient air.

Effective methods are available for control of chlorine emissions. No information has been found on the economic costs of chlorine air pollution or on the costs of its abatement.

Methods of analysis are available; however, they are not sufficiently sensitive or selective for determining atmospheric concentrations of chlorine.

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1. INTRODUCTION

Chlorine is a dense, greenish-yellow gas with a distinctive, irritating odor. It is noted for its very strong oxidizing and bleaching properties. Because of these properties, chlorine can be extremely hazardous to all life forms, as well as corrosive to metals and other materials. Although chlorine is not flammable, it can support combustion, and many materials (such as hydrogen) and many metals can burn in a chlorine atmosphere--sometimes with explosive violence (see Tables 1 and 2 in the Appendix for the reactivity and the physical properties of chlorine).

Several incidents of accidental chlorine leakage have led to injury and death of humans and animals, and damaged many species of plants.

The production of chlorine in the United States has doubled in the past 10 years and is projected to continue to increase at a rate of approximately 7 percent per year. The largest users of chlorine are the chemical industry and the pulp and paper industry. The major commercial source of chlorine is the electrolysis of alkali chloride solutions. The chlorine is usually liquefied before use or storage. The liquefication process can be an important source of chlorine atmospheric emissions if not carefully controlled. Effective methods are available for control of chlorine emissions.

2. EFFECTS

Chlorine gas is a very strong oxidizing agent, capable of reacting with organic as well as inorganic materials. This property makes it dangerous to humans, animals, plants, and numerous materials. Furthermore, in the presence of moisture chlorine reacts to form hypochlorite, another strong oxidizing agent (the active ingredient in liquid household bleach), and hydrochloric acid, one of the common strong acids. If these compounds are formed in the mucous membranes of the body from chlorine present in the atmosphere, damage to the tissues could result.

2.1 Effects on Humans

The sensitivity of humans to chlorine gas varies greatly among individuals.[52] The main effect which has been noted is the irritating and corrosive action on the mucosa of the eyes, nose, throat, and respiratory tract. Exposure to high concentrations of chlorine can damage the lungs and has resulted in pulmonary edema,[17,58,63,81] pneumonitis,[17,63,64] emphysema,[52,81] and bronchitis.[52,64,107] In extreme cases, the damage to the lungs may be severe enough to result in death by suffocation.[18,24] Available evidence suggests that humans can develop some tolerance to low concentrations of chlorine.[94]

2.1.1 Acute Effects

At chlorine gas concentrations of 3,000 $\mu g/m^3$ of air (1 ppm) or less, persons, particularly those not accustomed to

chlorine become disturbed and exhibit noticeable symptoms of irritation.[39,94] Depending on the individual's sensitivity, irritation may first be noted in the eyes, nose, or throat. (The more recent studies indicate that the odor threshold is also below 3,000 $\mu g/m^3$; see Section 2.1.3). Irritation of these areas will increase during the first hours of exposure. However, men may work without interruption in an environment at concentrations of chlorine of 3,000 to 6,000 $\mu g/m^3$.[52] Many reports indicate that persons habitually exposed to chlorine develop some degree of tolerance.[94] Dixon and Drew,[32] however, reported that there is no tolerance in men, but rather, that workers seem to be able to voluntarily reduce their respiratory tidal volume in the presence of chlorine. At any rate, Kramer[64] reports that workers who are continually exposed to chlorine are able to tolerate 15,000 to 25,000 $\mu g/m^3$ (5-8 ppm) for significantly long periods.

Normally, chlorine exposures of 9,000 to 18,000 $\mu g/m^3$ (3 to 6 ppm) cause a stinging or burning sensation to eyes, nose, and throat.[52] This is in contrast to earlier data of Fieldner et al.,[18,24] still generally used, which give the threshold concentration for throat irritation as approximately 45,000 $\mu g/m^3$ (15.1 ppm) and for coughing as approximately 90,000 $\mu g/m^3$ (30.2 ppm). Furthermore, a headache may develop, caused by irritation of the accessory nasal sinuses.[52]

Exposure of sufficient duration may cause redness and watering of eyes, sneezing, coughing, and huskiness or loss of voice. Moreover, bleeding from the nose may result, and sputum from the pharynx and trachea may contain blood. Muscular soreness, other than from extensive coughing, is absent. Bronchospasm of a relatively transient nature occurs in almost all chlorine exposures, including the minor exposures.[64]

Exposure to environments containing chlorine concentrations of 40,000 to 60,000 $\mu g/m^3$ (14 to 21 ppm) for 30 to 60 minutes is dangerous, while concentrations of 290,000 $\mu g/m^3$ (100 ppm) cannot be tolerated for more than one minute.[52]

Symptoms observed in persons following a heavy exposure to chlorine include choking, nausea, vomiting, retching, dyspnea, burning eyes, headache, dizziness, anxiety, and syncope.[17,58] Examinations after exposures reveal an increase in body temperature, anorexia, diffuse cracking rales, and acute conjunctival infection with profuse tearing and photophobia; muscular weakness and a decrease in stamina are also noted. Heavy exposure to chlorine may also lead to respiratory disorders (see Section 2.1.2).

Inhalation of massive doses of chlorine gas will result in destruction of tissues. If damage to lung tissue is extensive, death by suffocation soon occurs.[15] Moreover, with sudden extreme exposures, shock may occur, with a spontaneous constriction of the trachea or bronchi to a degree which causes suffocation. Deaths from chlorine inhalation will usually occur within minutes, or, at most, within several days.

Chlorine is very reactive with mucosa; therefore, the effects on the body are normally encountered only in a localized area. Hence, entrance of chlorine or its reaction products into the blood circulation occurs only from massive exposures (such as 2 g/m^3 of chlorine or 2,000 ppm, which is rapidly fatal).

A summary of the reported effects on man due to inhalation of chlorine gas is given in Table 3 in the Appendix.

2.1.2 Chronic Effects

Generally, it is reported that low concentrations of chlorine gas do not cause chronic effects. A common statement is that, based on examination of workers exposed daily to detectable concentrations of chlorine, there are no chronic systemic effects.[18,24] However, there is a paucity of data available on exposures to low concentrations over long periods of time. Recent studies indicate that some of the early data may be invalid, e.g., the odor threshold value has decreased by a factor of ten with recent data (see Section 2.1.3). Furthermore, some of the other physiological response data discussed in Section 2.1.1 suggest that the stated absence of chronic effects at low concentrations of chlorine needs further verification.

Patil et al.[90] studied 332 diaphragm cell workers exposed to chlorine. Time-weighted average (TWA) exposures to chlorine ranged from 18 to 4,260 $\mu g/m^3$ with a mean of 450± 870 $\mu g/m^3$. Significant correlations* were found between

*$p \leq .05$

exposure to chlorine and various effects, i.e., tooth decay, anxiety and dizziness, and leukocytosis. Hematocrit readings showed a significant inverse correlation with exposure. These investigators also noted that workers were in good health, and not affected in any clinically duplicable way by the years of continual exposure to low concentrations of chlorine. Acute exposure did cause temporary illness, but without evidence of permanent damage. In 1964, Pendergrass[94] reported that a study was in progress to determine the effect of long term exposure (several years) to low concentrations of chlorine ($< 3{,}000\ \mu g/m^3$).

Ronzani[100] in 1909 studied men working in bleaching rooms, exposed to chlorine concentrations of approximately $15{,}000\ \mu g/m^3$ of chlorine (5 ppm) in air. He found that these men aged prematurely, suffered from disease of the bronchi, and were predisposed to tuberculosis. In addition, he noted that their teeth were corroded from the hydrochloric acid produced by reaction of chlorine with the moisture in the mouth, and also observed inflammation or ulceration of the mucous membrane of the nose.

Recognition of the danger of continuous exposure to low concentrations is further supported by the results described by Skjanskaja and Rappoport.[110] These investigators exposed rabbits to concentrations of chlorine ranging from 2,000 to $5{,}000\ \mu g/m^3$ (0.7 to 1.7 ppm) over periods lasting up to 9 months. This produced a loss of weight and an increased

incidence of respiratory disease among the rabbits. Furthermore, other investigators have reported that in experiments with guinea pigs, small concentrations of chlorine accelerate the course of experimental tuberculosis.[4] These studies tend to disprove the earlier belief of Baskerville[8,9,10] that small amounts of chlorine in a working environment decrease the incidence of respiratory diseases among workers, possibly by sterilizing the air.

As a consequence of a heavy exposure to chlorine, a person may develop lung ailments, including pulmonary edema,[17,58,63,81] pneumonitis,[17,63,64] emphysema,[52,81] and bronchitis.[52,64,107] The common opinion in the literature is that complete recovery will generally occur rapidly with no further complications if the illness is not too severe. In addition, pulmonary damage may be incurred as a result of the exposure. Whether or not there is permanent pulmonary damage has been debated for many years in the literature. A summary of some of the reported studies and views are presented in the following paragraphs.

Berghoff,[12] after reviewing in 1919 the record of 2,000 men exposed to war gases, concluded that half of these men had evidence of emphysema or bronchitis, but he felt that they would regain normal respiratory functions. In contrast, Sandall,[105] who studied 83 gassed British pensioners, and Hankin and Klotz,[49] who studied 166 American veterans hospitalized from gassing, concluded in 1922 that the lung damage

would result in permanent disability. Pearce[91] found in 1920 that a person exposed to chlorine gas showed clinical evidence of obstructed airways and emphysema, 12 months after exposure. Gilchrist and Matz[45] in 1933 found that some veterans with a history of chlorine exposure had bronchitis and emphysema, or both; five had coexistent tuberculosis, which these authors felt was related to the chlorine gas exposure. However, the general attitude prevailing today is similar to that expressed by Penington in 1954.[93] He concluded, citing the earlier study (1938) of Price[78] for support, that comparatively few victims of war gas suffered permanent pulmonary injury.

Jones[57] in 1952 conducted follow-up studies of 820 industrial cases of chlorine poisoning and concluded that there was no clinical or radiological evidence of permanent lung damage. In earlier studies (1923), Haggard[48] suggested that permanent pulmonary damage and disability resulting from toxic gas inhalation might be undetectable by routine clinical and radiological studies.

Kowitz et al.[63] in 1967 studied 59 persons exposed to chlorine gas from a 1961 accident (see Section 2.1.5). These people were examined for 2 to 3 years after the accident. It was found that a decrease in diffusing capacity of the lungs had resulted from the exposure. Several of the subjects had mean respiratory function changes comparable to alveolo-capillary injury. Even after 2 to 3 years, a decrease in lung

volume and diffusing capacity remained. It was concluded by the authors that the chronic effects of chlorine poisoning are not clearly understood and, on the basis of cited animal studies, they suggested that clinically undetectable damage may result from exposures to chlorine gas or other gases.

Wells et al.[131] examined 12 persons exposed to chlorine as a result of the 1961 tank car accident described by Joyner and Durel in 1962[58] (see Section 2.1.5). These persons were examined for pulmonary function 3 years and/or 7 years after the acute exposure. Measurements included tests of residual volume, total lung capacity, pulmonary diffusing capacity, and vital capacity. The authors concluded that "No appreciable physiologic disorders were detected that could not be explained by associated clinical studies."

2.1.3 Sensory Thresholds

A frequently cited reference gives approximately 10,000 $\mu g/m^3$ as the odor threshold for chlorine.[39] However, more recent studies indicate the threshold to be much lower. These results indicate that the odor threshold is below 1,000 $\mu g/m^3$, as shown by a summary of some of the published data which propose values of 150,[11] 750,[112] 800,[120] and 940 $\mu g/m^3$.[114] Beck,[11] as cited in another paper,[94] found that odor threshold values are higher if the emission of the gas takes place very gradually.

Takhirov[120] studied sensory thresholds for the eye reflex and "respiratory movement." The results indicate that

the threshold for chlorine effect on eye sensitivity to light was the same as for the odor threshold: namely, 800 $\mu g/m^3$. However, in optical chronaxy studies, a concentration level of 1,500 $\mu g/m^3$ of chlorine was necessary to elicit a reflex response. This was also found to be the case with reflex activity changes in the rhythm and amplitude of "respiratory movements."

2.1.4 Synergistic Effects

Stayzhkin[112] in 1962 cited the study of Shtessel, who investigated the combined effect on animals of chlorine and sulfur anhydride as well as of chlorine and nitrogen oxides. He initially established the lethal dose for cats and mice for each of these gases separately, and then the lethal dose for certain combinations of these gases. The results indicated that inhalation of chlorine and nitrogen oxides in lethal doses produces similar effects as found when administered separately. However, when concentrations of chlorine and of sulfur anhydride that usually produce lethal effects were combined, the results were weaker than when either concentration was given alone; this suggested a slight antagonistic action of the combination.

Stayzhkin[112] investigated the effect of chlorine and hydrogen chloride gas mixtures on man. In his study of the odor threshold, the effect on reflex reactions of eye sensitivity to light and of optical chronaxy were examined. His results indicated that the two gases acted in combination

(in an additive manner) to produce a perceivable odor when neither gas could be detected alone. The effect is expressed by the following relationship where X must equal one or greater to have a perceivable odor:

$$X = \frac{\text{Concentration } Cl_2}{\text{Odor threshold } Cl_2} + \frac{\text{Concentration HCl}}{\text{Odor threshold HCl}}$$

Furthermore, the physiological and neurological effects of the mixtures were also additive.

2.1.5 Chlorine Gas Exposure to Communities Through Accidents

In March 1961, while a freighter was unloading supposedly empty liquid chlorine cylinders in the harbor of Baltimore, the main valve of a cylinder that was being hoisted from the ship's hold snapped off.[63] No data were given regarding the amount of chlorine that was in the cylinder before it ruptured. As a result of this accident, 156 persons were examined at three hospitals and 37 were given further treatment. Several men returned to the hospital within 48 hours and were subsequently admitted for treatment. Of the 17 persons admitted at one hospital, 11 had respiratory distress: four, hemoptysis; eight, rales; six, wheezes or rhonchi, or both; and four, edema or infiltrate. One of these patients developed bacterial pneumonia. A follow-up examination of these 17 patients was given at 30 to 60 days, 6 months, and 14 months, as well as 2 to 3 years after the accident. It was found that 19 to 35

months after the exposure, the victims generally complained of shortness of breath and fatigue on exertion. Other symptoms noted, in order of descending frequency, were: cough, nonspecific chest pain, frequent headaches, dryness of the oropharyngeal membranes, decreased stamina, and muscular weakness. Eleven of the 17 victims had mean respiratory function changes comparable with those resulting from an alveolocapillary injury. During the 2 to 3 years of the survey, continuing repair with increased airway resistance was noted. A decrease in lung volume and diffusion capacity remained after the completion of the survey. An additional study of 59 of the 156 persons exposed to the chlorine, including the 11 mentioned above, was made 11 to 20 months after the accident. The conclusion from this follow-up study was that a decrease in vital capacity, decreased pulmonary elasticity, and a decrease in diffusion capacity resulted from the exposure to chlorine.

In January 1961, a tank car was derailed at the rural community of La Barre, La., spilling 6,000 gallons (36 tons) of liquid chlorine.[58] The cloud of chlorine gas that formed covered approximately 6 square miles, necessitating the evacuation of some 1,000 people. Air samples taken in the area 7 hours later showed that chlorine concentration ranged from 1,200,000 $\mu g/m^3$ (400 ppm) 75 yards from the site of the accident to 30,000 $\mu g/m^3$ (10 ppm) in the fringe areas of the cloud.

Approximately 100 people were treated for varying degrees of chlorine exposure, and an 11-month-old infant died as a result of the exposure to chlorine. Of the 65 persons checked at one hospital, 15 were admitted for further treatment. Patients complained of burning eyes and evidenced acute conjunctival infection with lacrimation and photophobia. Those persons heavily exposed experienced severe dyspnea, coughing, retching, and vomiting. Increased body temperatures (reaching 101°F), anorexia, and moist rales were common. Of the original 15 persons admitted to the hospital, 10 developed unmistakable pulmonary edema. By the 16th day after admittance, all the patients had been discharged. In addition to the humans affected, many domestic and commercial animals located within 1½ miles downwind of the accident were killed.

In 1947 in Brooklyn, N.Y., gas escaped near a subway ventilator grating after a 100-pound tank of liquid chlorine developed a 1/8-inch hole.[17] The people in the area were exposed for approximately 17 minutes before the chlorine emissions could be stopped. Those exposed were overcome by choking, nausea, vomiting, anxiety, and syncope. Some were lying prostrate in the street, while many others demonstrated distress and weakness. People with mild exposures mainly exhibited burning of eyes and nose, lacrimation, and rhinorrhea. Of the 418 people examined, 208 were admitted to hospitals for treatment. At one hospital all of the 33 patients treated had

varying degrees of tracheobronchitis; 14 developed pneumonia, and 23 developed pulmonary edema. After hospitalization, further observations were made of 29 of the patients. Sixteen of these showed anxiety reactions with phobias, hysterical phenomena, and psychosomatic dysfunction for periods lasting from 1 to 16 months. However, none of the 29 patients developed permanent pulmonary disease.

2.2 Effects on Animals

2.2.1 Commercial and Domestic Animals

No information has been found in the literature pertaining to injury of domestic, commercial, or wild animals as a result of exposure to chlorine gas either in ambient air or near plants that produce or use chlorine. Several animals including dogs, cats, horses, mules, chickens, hogs, cows, and ducks died as a result of an accidental spillage of chlorine in La Barre, La.[58] (see discussion in Section 2.1.5).

2.2.2 Experimental Animals

Numerous animal studies on the effects of chlorine have been reported and are summarized in Table 4 in the Appendix. The results of several reports have been discussed in Section 2.1. Autopsy data on mice and rats exposed to chlorine concentrations of approximately 3,000,000, 750,000, and 189,000 $\mu g/m^3$ are given in Table 5 in the Appendix. These autopsies[130] indicate that the rats showed much edema and slight to

moderate hemorrhage of the lungs, after exposure, whereas the mice showed somewhat less edema and more hemorrhage. Furthermore, chlorine appears to produce greater amounts of lung hemorrhage and edema than the same concentrations of ammonia, hydrogen cyanide, sulfur dioxide, and hydrogen sulfide.

2.3 Effects on Plants

2.3.1 Phytotoxicity

Brennan, Leone, and Daines[13] studied the effects of various concentrations, 300 to 4,500 $\mu g/m^3$ (0.1 to 1.5 ppm) of chlorine gas on 26 different species of plants. The most common symptoms of chlorine poisoning were necrosis and bleaching of the foliage, which occurred within a day or two after the chlorine exposure. Bleaching of the leaves was a typical symptom which developed from exposure to low concentrations of chlorine. Such species as spinach and cucumber had a bleaching pattern similar to that produced by ozone. In respect to necrotic tissue, the leaf color ranged from white to tan to brown. Generally, the necrotic area was marginal and interveinal. However, there are many exceptions: for example, tomato, tobacco, radish, and cucumber had necrotic areas scattered over the leaf blade, while alfalfa in some cases had necrosis only of the veins. The oldest and the middle-aged leaves appeared to be more susceptible to chlorine injuries than the younger leaves. Furthermore, the

cotyledons showed greater resistance to injury than the mature primary leaves. The upper leaf surface appeared more sensitive to chlorine gas than the bottom, although injury occurred on both sides.

Zimmerman[137,138] found similar responses in 16 species of plants exposed to chlorine concentrations of 1,380 to 14,010 $\mu g/m^3$ (0.46 to 4.67 ppm). The most characteristic symptom was spotting of the leaves similar to that observed with sulfur dioxide. The spots initially appeared as cooked areas that turned straw-yellow or brown within a few days. Leaf fall was associated with moderate to severe injury.

Other symptoms also found include epinasty of tomato leaves at chlorine concentrations insufficient to produce spots, and "cupping" of the younger leaves of squash.[13] In some species, e.g. sugar beets, the upper epidermis may be injured, giving it a detached "silver-leaf" appearance.[121]

It was reported that leaf fall may occur without the evidence of symptoms.[113]

Heck et al.[39] exposed plants to 62 ppm of chlorine, and noted that squash, soybean, and cowpea plants showed a loss of turgor in addition to the necrosis. After exposing tomato, buckwheat, and tobacco to chlorine concentrations of 3,000 $\mu g/m^3$ (1,000 ppm), Thornton and Setterstron[123] observed that the stem tissue was bleached and had a cooked appearance.

2.3.2 Sensitivity of Plants

The sensitivity to chlorine gas varies greatly among

different species of plants.[13,138] Some of the most susceptible to injury from chlorine gas include radish, alfalfa, peach, coleus, cosmos, buckwheat, and tea rose. Species that have a high resistance include Chinese holly, eggplant, tobacco, Werech enopodium, polygonum, oxalis, begonia, and pepper. Table 6 in the Appendix summarizes the effects of chlorine gas on various plants as reported in the literature.

Thornton and Setterstrom[123] compared the toxicity of five gases and concluded the relative toxicity to green plants as $Cl_2 > SO_2 > NH_3 > HCN > H_2S$. Zimmerman[137] arrived at a similar conclusion, namely: $HF > Cl_2 > SO_2 > NH_3 > H_2S$. Brennan et al.[13] agree that chlorine is a stronger phytotoxicant than sulfur dioxide. McCallan and Setterstrom[79] reported the same results as Thornton and Setterstrom.[123] Moreover, they compared the overall effect of chlorine on other organisms and concluded that the order of toxicity susceptibility was as follows: leaves>fungi, bacteria, stems, animals>seeds and sclerotia.

2.3.3 Effect of Moisture

Tomato plants were exposed to chlorine concentrations of 930, 1,830, and 4,140 $\mu g/m^3$ for 2 hours. During the exposure some plants were periodically sprayed with water, whereas others were not. When the plants sprayed with water and those left unsprayed were compared, no difference was found in the degree of injury resulting from the exposure to chlorine.[13]

Barton[7] investigated the effects of chlorine gas at concentrations of 750,000 and 3,000,000 $\mu g/m^3$ (250 ppm and

1,000 ppm) on the germination of dry and soaked radish and rye seeds. Dry radish seeds did not appear to be affected by a 960-minute exposure at either concentration level. However, after 960 minutes of exposure to 3,000 $\mu g/m^3$ (1,000 ppm) chlorine, the soaked seeds showed approximately 90 percent less germination, and approximately 16 percent less at 750,000 $\mu g/m^3$ (250 ppm) chlorine. Germination of the dry rye seeds showed a reduction of approximately 50 percent at the higher chlorine concentration for 960 minutes, while only slight reduction (approximately 10 percent) at the lower concentration. The soaked rye seeds started to show a reduction in germination at 240 minutes when exposed to 750,000 $\mu g/m^3$ (250 ppm) chlorine, and at 60 minutes with 3,000,000 $\mu g/m^3$ (1,000 ppm). When exposed for 960 minutes, there was a 68 percent reduction at the lower concentration, whereas at the higher concentration there was no germination at all.

2.3.4 Effect of Light

Experiments were performed to determine the effect of light on the response of tomato plants to chlorine gas.[13] Plants kept in the dark for 20 hours prior to fumigation with 1,800 $\mu g/m^3$ (0.60 ppm) of chlorine for 2 hours were injured to the same extent as plants kept under the usual greenhouse conditions prior to similar exposure. However, plants kept in the dark for 20 hours after the exposure to chlorine had 50 percent less damage than those plants treated under similar

conditions, but kept in the daylight in a greenhouse after exposure.

Thornton and Setterstrom[123] compared the effect of clear as compared to cloudy weather. Tomato plants were exposed to chlorine concentrations ranging from 1,200 to 3,000,000 $\mu g/m^3$ (4 to 1,000 ppm) for periods of as long as 2 hours. The results indicated that the injury to the leaf tissue occurs in a shorter period of time on a clear day than on a cloudy day, particularly at low concentrations of chlorine.

2.3.5 Effect of Water Stress

Brennan, Leone, and Daines[13] investigated the effect of water stress on tomato plants exposed to a chlorine concentration of 1,800 $\mu g/m^3$ (0.60 ppm) for 2 hours. The turgid plants had necrotic injury on both the oldest and the middle-aged leaves, while only 25 percent of the wilted plants that were tested developed injury, which was slight. Spraying of the plants with N-dimethylamine to close the stomata also reduced the injury from chlorine, but not as effectively as the reduction produced by wilting.

Zimmerman[138] similarly showed that wilted plants have a pronounced resistance to chlorine as compared with turgid plants.

2.3.6 Plant Accumulations

Brennan et al.[13] investigated the relationship between degree of tomato plant injury and the chloride content of the leaves. Tomato plants were fumigated with 1,200 $\mu g/m^3$ of

chlorine (0.40 ppm) for 6 hours, which caused no visible foliage injury; chlorine concentration of 1,800 $\mu g/m^3$ (0.60 ppm) for 4 hours, which caused moderate injury; and concentrations of 5,200 $\mu g/m^3$ (1.40 ppm), which caused severe injury. Analysis of the leaves indicated that the chloride content of the tissue did not correlate either with the level of chlorine exposure nor with the amount of injury to the plant. To substantiate these results the chloride content of the leaves (top and lower), stems (top and lower), and petioles (top and lower) were analyzed before and after exposure to 1,800 $\mu g/m^3$ (0.60 ppm) of chlorine for 3 hours. The chloride distribution of these six fractions did not appear to significantly change from an analysis made immediately after exposure to an analysis made 1 day later. Hence, it was concluded that chloride analysis of plants cannot be used as a method of diagnosing injury that results from chlorine gas exposure.

However, Liegel and Oelschlager[72] observed an increase in the chlorine content of lettuce and spinach plants after fumigation with chlorine gas. Sufficient data were not given (e.g., the chlorine concentration used) to compare their results with those of other studies.

An incident occurred in which silver maple trees were damaged as a result of emissions (from a glass manufacturing company) containing both hydrogen chloride and some chlorine. Analysis indicated a slight increase in chloride in exposed

maples as compared to uninjured maples.

A study of the effects of gases on the pH of the leaves and stems of tomato plants was reported by Thornton and Setterstrom.[123] Chlorine was found to be more effective in lowering the pH of the leaves than was ammonia, sulfur dioxide, hydrogen cyanide, or hydrogen sulfide. Plants exposed for 15 minutes to chlorine concentrations of 12,000 $\mu g/m^3$ (4 ppm) reduced the pH by 0.3 units, while 960 minutes at the same concentration reduced the pH by 1.0. After 960 minutes at 3,000 $\mu g/m^3$ (1 ppm) chlorine, the pH was lowered by 0.2; at 48,000 $\mu g/m^3$ (16 ppm), by 1.6; at 189,000 $\mu g/m^3$ (63 ppm), by 4.7; at 750,000 $\mu g/m^3$ (250 ppm), by 4.5; and at 3,000,000 $\mu g/m^3$ (1,000 ppm), by 5.5. In contrast, the pH of the stem exhibited little change; at 3,000,000 $\mu g/m^3$ (1,000 ppm) of chlorine for 960 minutes, the pH was lower by only 1.0 unit. Additional tests indicated that the older and lowermost leaves showed the greatest pH change, while pH change of the stems was uniform from top to bottom. Furthermore, it was also found that the effectiveness of chlorine in lowering the pH was much greater in sunlight than in darkness. Thus, exposure to 3,000,000 $\mu g/m^3$ (1,000 ppm) of chlorine for 4 minutes decreased the pH in the leaf tissues by approximately 2.8 units in sunlight, but only approximately 0.3 units in darkness. Under the same conditions, the pH of the stems was reduced by approximately 0.2 units in sunlight and approximately 0.05 units in darkness.

The results of these studies suggest that the amount of plant injury was positively correlated with the effectiveness of chlorine (as well as of sulfur dioxide, ammonia, and hydrogen sulfide) in causing a change in pH.

2.3.7 Episodes of Plant Damage

Several cases have been reported in the literature in which plants have been injured as a result of accidental chlorine leaks around sewage treatment plants, factories, and even swimming pools.[53,86,106,116,137,138] However, no chlorine air measurements were given in these reports.

Stout[116] in 1932 reported an instance in which lettuce and weeds growing near a sewage treatment site in California were injured as a result of a chlorine leak from the sewage chlorinating apparatus.

Zimmerman[137,138] later reported two such incidents in the vicinity of Yonkers, N.Y. One occurred near a swimming pool as a result of a leak from a cylinder of chlorine which was used to purify the water. The other involved the accidental emission of chlorine gas from a factory. As a consequence of these two episodes, some 30 species of plants were injured, including Ailanthus sp., apply (Malus), cherry (Prunus sp.), maple (Acer sp.), smartweed (Polygonum sp.), Weigela sp., basswood (Tilia americana L.), dogwood (Cornus sp.), elm (Ulmus sp.), ash (Fraximus sp.), sweet gum (Liquidambar sp.), hemlock (Tsuga sp.), oak (Quercus sp.), and white pine (Pinus alba).

Schmidt[106] reported that an accident with chlorine caused defoliation of peach, apple, and quince trees.

Hindawi[53] mentioned an accidental chlorine release from an industrial plant in a residential area in Cincinnati, Ohio. One day after the accident, leaves began to fall from tomato plants and trees in the area, and the silver maple trees showed marks similar to those caused by sulfur dioxide. Furthermore, privet hedges in the area were nearly bare.

Emissions from a glass manufacturing factory in another region injured shrubs, trees, and ornamental plants in the surrounding area. Specimens which were severely injured included maple, cherry, rose (both bud and bush), and begonia. Factory stack emissions analyzed after the episode contained 119 to 473 ppm of hydrochloric acid and 0.52 to 0.92 ppm of chlorine. Analysis of injured silver maples showed 4,700 ppm chloride compared to 3,800 ppm for the uninjured trees.

2.4 Effects on Materials

Examples in which chlorine gas has caused economic damage to material have not been discussed in the literature reviewed. However, the high reactiveness of chlorine with almost all metals (including iron, zinc, tin, silver, and copper) as well as with nonmetals (including most organic compounds) suggests that chlorine in sufficiently high concentrations would corrode metals, discolor and damage painted materials, and damage textile fibers.

Chiarenzelli and Joba[20] studied the corrosion of different metals in areas which have significant levels of atmospheric pollution. At one site, a heavy and unique tarnish film that was noted on silver was thought to be a result of the high chlorine concentrations in that area.

2.5 Environmental Air Standards

The American Conference of Governmental Industrial Hygienists (ACGIH) has adopted an 8-hour threshold limit value (TLV) for chlorine of 3,000 $\mu g/m^3$ (1 ppm).[124] This TLV is based on data which indicate that men can work at this concentration of chlorine without interruption from eye, nose, and throat irritation.[33]

West Germany has established the same TLV for working conditions as the United States (3,000 $\mu g/m^3$ or 1 ppm).[94] The West Germany Verein Deutscher Ingineure (VDI) Committee on Air Purification in 1960 established a chlorine "continuous exposure value" of 300 $\mu g/m^3$ (0.1 ppm) as a mean value for 30 minutes.[94] It is permissible to exceed this value, but for no more than three times in one day and by no higher than a 30-minute mean chlorine value of 1,500 $\mu g/m^3$ (0.5 ppm).

Russia has established a 24-hour average maximum allowable concentration of 30 $\mu g/m^3$ (0.01 ppm) of air for chlorine.[78] A single measurement should not exceed 100 $\mu g/m^3$ (0.033 ppm). Russia's maximum 8-hour occupational exposure for chlorine is 1,000 $\mu g/m^3$ (0.33 ppm), or one-third that adopted by the ACGIH.

3. SOURCES

3.1 Natural Occurrence

Natural occurrences of free chlorine gas are extremely rare, due to the high reactivity of chlorine with many substances. Volcanic gases contain very small amounts of chlorine gas.[22] Low concentrations of chlorine may, however, be formed by atmospheric reactions.[7] For example, chloride compounds and nitrogen dioxide may react to form nitrosyl chloride, which can decompose photochemically to yield free chlorine. Also, chlorides in the presence of strong oxidants (such as ozone) may be oxidized to chlorine.

3.2 Production Sources

The ever-increasing demand for chlorine has resulted in a doubling of production in the last 10 years (see Table 7 in the Appendix). In 1967, the production was approximately 7.65 million short tons.[29] The projected production for 1971 is 10 million short tons, assuming a rate of increase in production of 7 percent per year, which is similar to the rate of the past 10 years.[23]

The major processes for the production of chlorine in the United States are the electrolysis of aqueous alkali chloride via the diaphragm cell or mercury cell, and to a lesser extent, fusion electrolysis of alkali chlorides (Down Process), and the nitric acid process. Diaphragm - and mercury - cell processes accounted for over 95 percent of

the chlorine produced in 1964. A more detailed description of these processes can be found in several reference sources.[5,22,75,108,115]

There were 69 plants producing chlorine in the United States in 1964.[21] A list of the major producers is given in Table 8 in the Appendix. A more detailed list is to be published of producers in the United States, giving the type of process used, nominal capacity, and location.[5]

The major sources of atmospheric emissions of chlorine from the production processes are the following: liquefication processes, the filling of containers or transfer of liquid chlorine from one container to another, the cleaning of returned tank cars containing residual chlorine, the improper treating of spent brine solution and of "sniff" or "blow" gas (the gas remaining after the final liquefication step), and occasional equipment failure (for example, a chlorine compressor breakdown).[11,15]

3.2.1 Electrolytic Diaphragm Cells

Electrolytic diaphragm cells account for over two-thirds of the chlorine production.[5] In this process, a saturated aqueous solution of sodium chloride or potassium chloride is electrolyzed. Chlorine gas (98 percent chlorine) is liberated at the anode, while alkali solution, as well as some by-product hydrogen gas, is liberated at the cathode.[5,75] The chlorine gas leaves the cell and is passed through coolers. The chlorine-

saturated water that condenses in the coolers passes to the sewer through a limestone pit and is not considered to be a source of air pollution.[75] The partly dried chlorine gas from the coolers is further dried by contact with sulfuric acid. The spent sulfuric acid is either reclaimed or discharged to the sewer after neutralization.

The dried chlorine gas can be used directly, but it is more common to liquefy the gas by compression and cooling. The gas residue after the final liquefication process, called "sniff gas," "blow gas," "vent gas," or "tail gas," consists of air and chlorine. This mixture normally consists of about 20 to 50 percent chlorine, or as much as 8 percent of the plant's total chlorine production.[119] Thus, if the sniff gas were allowed to be directly vented to the atmosphere, 1 to 6 tons of chlorine would be emitted for every 125 tons produced.[55] The filling of tank cars, barges, and other containers with liquid chlorine can result in a loss of 2 percent of the chlorine production.[42] In many plants, this vent gas is collected and piped either for direct use without further purification, or to recovery systems.[115] The sniff gas is used by other in-plant processes (such as preparation of bleaching solution and chlorination of organic compounds), or is treated further by either scrubbing or absorption methods to remove the chlorine (see Section 4). In most cases, the chlorine can be recovered economically by means of these purification methods.

3.2.2 Electrolytic Mercury Cells

Electrolytic mercury cells account for about one-fourth of the total chlorine production.[5] To produce these cells, solution of sodium or potassium chloride is electrolyzed. Chlorine gas collects at the anode, while at the mercury cathode sodium or potassium metal is produced, which forms an amalgam with the mercury. The amalgam is then reacted with water in another section to yield alkali hydroxide and hydrogen gas. The advantage of this process over the other electrolytic processes is the high purity of alkali hydroxide which is produced.

The drying, liquefication, and collection of the chlorine gas is similar to that discussed for the diaphragm-cell process.

3.2.3 Fusion Electrolysis of Chloride Salts

The fusion electrolysis of chloride salt accounts for less than 5 percent of the total chlorine production.[5] This method is primarily used to prepare pure metals, namely magnesium and sodium metal, from their chloride salts.[75] The co-product--chlorine gas--is collected at the anode and further processed by a method similar to that described for the diaphragm-cell process.

3.2.4 Other Processes

Other processes that have been used for making chlorine gas include (1) treatment of sodium chloride with nitric acid,

(2) electrolysis of hydrochloric acid, and (3) oxidation of hydrochloric acid with catalyzers.[75,99] Of these, only the first is of importance in the United States, but even so, this process accounts for less than 1 percent of the present United States production.[5]

3.3 Product Sources

The largest consumer of chlorine is the chemical industry, which uses this substance for the manufacture or preparation of chemical compounds--both organic and inorganic. Nearly 80 percent of the total chlorine production of this country is consumed in this way, with the organic compounds accounting for the greater part--approximately 70 percent of the total production.[5] The second largest user is the pulp and paper industry (which consumes approximately 16 percent of total production), followed by water and sewage treatment (approximately 4 percent of total production).[5] Table 9 in the Appendix gives the consumption of chlorine by uses for 1963 and 1964.

3.3.1 Chlorinated Organic Chemicals

The manufacturing of chlorinated organic chemicals constitutes the largest use of chlorine by the chemical industry. Uses include the preparation of:

Solvents: trichloroethylene, perchloroethylene, methylene chloride, and carbon tetrachloride;

Plastics and fibers: vinyl chloride and vinylidene chloride;

Pesticides and herbicides: DDT (dichloro-diphenyl-trichloro-ethane), benzene hexachloride, and toxaphene;

Refrigerants and propellants: freons and genetrons.

3.3.2 Other Organic Chemicals

Several chemicals are prepared via chlorination (dry or in the presence of water), followed by dehalogenation, to yield compounds that contain no chlorine, even though chlorine is used in their manufacture. These chemicals include ethylene glycols, glycerine, ethylene oxide tetraethyl and tetramethyl, lead additives, cellophane, pharmaceuticals, and detergents.

3.3.3 Inorganic Chemicals

A large variety of inorganic chemicals are made by using chlorine, including chloride salts (ammonium, calcium, and ferric chlorides, etc.), metals (e.g., aluminum), and other compounds (bromine, boric acid, paint coatings, silicates, phosphates, etc.).

3.3.4 Other Uses

A large amount of chlorine is being used by the pulp and paper industry, particularly for the oxidation of the odorous sulfur compounds present in the black liquor and in the bleaching operation.[25,133] The amount of chlorine needed to oxidize the sulfur compounds depends on the pH of the solution and the amount and type of sulfur compounds present. For example, to completely oxidize a mole of mercaptan in

neutral or acid conditions requires between 2.4 to 3.3 moles of chlorine, while under basic conditions, only 1.5 to 2.6 moles of chlorine are required. Sulfides require 2 to 3.4 moles of chlorine per mole of sulfide, depending on the conditions of the oxidation, while a mole of disulfide requires about 5.2 moles of chlorine.

Other uses include the production of compounds for household bleaches as well as for textile bleaching and finishing, water and sewage treatment, petroleum production and refining, rubber reclaiming, and food processes.

3.4 Environmental Air Concentrations

No information has been found on the environmental air concentrations of chlorine gas.

4. ABATEMENT

In plants producing chlorine gas, the electrolytic cells, the chlorine coolers, and the drying systems are operated at a slight vacuum, which normally prevents the emission of chlorine from these systems. In spite of this precaution, an upset or malfunction of the pumps or a large leak in the system may cause a temporary discharge of chlorine to the atmosphere.

The major source of chlorine emissions, however, is the presence of residual gases remaining after the liquefication of the chlorine cell gas. These residual gases may contain from 20 to 50 percent chlorine by volume, depending on the conditions of liquefication. Other sources of emission include the loading and cleaning of tank cars, barges, or cylinders; dechlorination of spent brine solutions; and power or equipment failures.[5,75,115] To prevent air pollution from these sources, the emissions from these systems can often be piped to the sniff gas system or tied in directly either to a scrubber system or a high stack for dispersion. When scrubbers are used, the effluent from the scrubber is vented to a tall stack.

Several efficient methods have been developed to control chlorine emissions.[5,99,115] The most common of these methods include the use of liquid scrubbers which employ either water,[14,42,55] alkali solutions,[16,99] carbon tetrachloride,[56,

[87,119,42] or brine solution,[51] as well as solid absorbents such as silica gel.[134] With the exception of the alkali solution technique, all these methods can be used to recover the chlorine. Other control methods which have been reported use sulfur dichloride,[59] hexachlorobutadiene,[108] and stannic chloride.[56]

Some producers send the untreated emission gases containing chlorine to other in-plant operations, where they are used directly, for such operations as chlorination of hydrocarbons.[5]

4.1 Water Scrubbers

When controlled by water scrubbers, the chlorine-containing vent gas is passed countercurrently to a water stream in a tower filled with ceramic packing. Upon treatment with a water scrubber, a vent gas initially containing 15 percent chlorine by volume yields an effluent gas containing 15,000,000 to 30,000,000 $\mu g/m^3$ (5,000 to 10,000 ppm) of chlorine.[14] It is common practice to pass gases from the water scrubber through the more efficient alkali scrubbers, or to tall stacks for dispersion. The chlorine-rich scrubber solution is heated so that the chlorine may be stripped and recovered. An alternate method of treatment of the chlorine-rich water is to pass the water over activated charcoal or iron filings. The result is an oxidation-reduction reaction that converts the chlorine to the noninjurious chloride ion.

4.2 Alkali Scrubbers

Contact of chlorine with alkali solutions (usually caustic or lime solutions) produces an effluent gas with a lower residual chlorine concentration (often below the odor threshold of about 1 ppm) than can readily be attained by water scrubbing.[5] The reaction products are bleach, salt, and water. The main disadvantage of this method is the cost as well as the difficulty of disposing of the bleach solution (hypochlorite). This bleach solution is sometimes reused by local plants (such as pulp and paper industries), or can be treated with carbon to reduce the chlorine to chloride ion. However, some producers dispose of the solution by dumping it in rivers and streams.

4.3 Carbon Tetrachloride Scrubbers

The advantage of using carbon tetrachloride as an absorbent is that its absorbing capacity for chlorine gas is 10 to 12 times greater than water, and the recovery of the chlorine much more complete.[115] However, losses of carbon tetrachloride have been reported as high as 30 pounds per ton of recovered chlorine.[87]

5. ECONOMICS

No information has been found on the economic costs of chlorine air pollution. Localized accidental emissions of chlorine could have an economic impact due to injury or death of humans, animals, and plants. Several instances of such episodes have been reported.

No information was found on the costs for abatement of chlorine air pollution. However, many companies recover the chlorine from gas streams for reuse or sale.

The production and consumption of chlorine have been discussed in Section 3. The production is expected to continue to increase for the next few years at the current rate of 7 percent. If this rate continues for 10 years, the production will double by 1978.

6. METHODS OF ANALYSIS

There appears to be no specific method for the analysis of chlorine. Most of the methods used rely on the oxidizing property of chlorine, and thus the presence of other oxidizing agents--such as ozone, bromine, nitrogen oxides, sulfur oxides, etc.--can sometimes seriously interfere with the analysis.

6.1 Sampling Methods

Chlorine gas samples are collected either in impingers containing a reactive liquid solution or on a solid which is impregnated with a reactive substance.

The common liquid solution used in the United States is dilute sodium hydroxide solution (approximately 0.01 to 0.1 N).[31,114] This basic solution converts the chlorine gas to equal amounts of chloride ions and hypochlorite ions. When the solution is made acidic, the reaction is reversed and the chlorine gas is regenerated. However, the hypochlorite ion can further slowly decompose (under basic conditions) to give more chloride ions and oxygen. The amount of hypochlorite ion that decomposes will result in a corresponding loss in chlorine gas when the solution is acidified.

Other common absorber solutions react with chlorine gas to directly or indirectly produce a color change in the solution, which is taken as an indication of the amount of chlorine in the sampled air or gas. A variety of solutions are used, which are discussed in the following sections.

The chlorine gas can also be absorbed on solids containing reactive substances similar to those used with the absorber solutions. A color change results which indicates the amount of chlorine that is present. The solid support is usually either paper or silica gel. The former is generally used for both qualitative and quantitative measurements, while the latter--found in the commercially available "detector tubes"--is usually reserved for quantitative measurement. Some of the reactive substances are discussed in the following section.

6.2 Qualitative and Semiquantitative Methods

Many types of chlorine-indicator papers have been described in the literature. Among the most common are the starch-iodide papers.[46,65,94,129] The basic reaction of chlorine gas with the potassium iodide of the paper yields free iodine, which then reacts with the starch to produce a blue color. (However, other oxidants can also turn the paper blue.) The limit of detection is approximately 2 to 6 ppm. A starch-iodide paper also coated with glycerin and sulfurous acid is reported to turn brown or black and to have a sensitivity of 0.25 to 12 ppm.[109] A buffered cadmium-iodide-starch paper is reported to be free of interference from nitric oxide.[117] Other common papers used include bromide-fluorescein papers,[46,67,69,136] which change from yellow to red or rose, with a sensitivity of approximately 10 ppm; and o-tolidine

papers,[26,46,69,117] which turn yellow or bluish green (depending on other reagents present), with a sensitivity of approximately 2 ppm. Other indicator papers that have been reported include dimethylaniline,[129] aniline,[27,46,71,135] o-phenetidine,[88] and benzidine.[102]

Rapid semi-quantitative determination of chlorine gas can be made with commercially available gas-detecting tubes.[38,66,89] These tubes contain a solid-coated reactive material which changes colorimetrically when exposed to a specific gas or to certain types of gases. A given volume of the gas sample is passed over the absorbent, and the amount of absorbent that changes color (measured by the length that is affected) is used to determine the amount of particular gas being tested (in this case, chlorine gas). Reactive substances that have been used as indicators for chlorine are o-tolidine,[62] bromide-fluorescein,[30,37] and tetraphenylbenzidine.[80]

Continuous sampling instruments for detection of chlorine gas have been developed, based on the reflectance from a chlorine-sensitive coated paper.[2,61,118] An alarm system can be used with the instruments to warn when a certain limit has been reached. Sensitivity ranges from 300 to 9,000 $\mu g/m^3$ (0.1 to 3 ppm) of chlorine.

6.3 Quantitative Methods

Most quantitative methods of analysis for chlorine gas are based on colorimetric reactions. A sensitive reagent

commonly used is o-tolidine.[44,84,92,97,114,128] The air sample can either be passed directly into an acid solution of o-tolidine or collected in dilute sodium hydroxide, which can later be acidified and the o-tolidine then added. The latter method has the advantage that the time allowed for the development of color can be controlled and maximized. Acidification yields a more stable yellow-to-orange color. Color comparisons can be made with standardized color solutions by visual methods or in a spectrophotometer at 435 and 490 mμ. Approximately 3 liters of air must be sampled to detect chlorine concentrations of 3,000 $\mu g/m^3$. This method is reported to be better than 99 percent efficient. However, the presence of other oxidants--such as chlorine dioxide, ozone, ferric and manganic compounds, and nitrates--may interfere with this method.

In a new method used in England,[85] the reagent 3,3'-dimethylnaphthidine turns mauve in the presence of chlorine gas. This reagent is about eight times more sensitive than o-tolidine. Other oxidizing agents, such as bromine, chlorine dioxide, nitrogen dioxide, etc., interfere with the test.

One reagent is reported to be specific for chlorine gas, with a sensitivity of 1,000 $\mu g/m^3$, even in the presence of such oxidants as bromine, ozone, and nitrogen oxide.[30] The method is based on the oxidation of arsenious anhydride in an alkaline solution in the presence of potassium iodide and starch. The solution turns blue in the presence of chlorine.

Other reagents used for determination of chlorine gas that have been reported in the literature include: dimethyl-p-phenylenediamine,[1,95] methyl orange,[65,103,120] iodide-starch,[30] iodide-starch-arsenic oxide,[74,96] benzidine acetate,[30] bromide-fluorescein,[30] resorufin,[34] 4,4'-tetramethyldramino-diphenylmethane,[76] and rosaniline hydrochloride.[73]

Recently an air analyzer for chlorine gas was reported, based on polarographic measurements with a special galvanic cell.[35]

REFERENCES

1. Alfthan, K., and A. C. Jarvis, New Indicator for Chlorine, J. Am. Water Works Assoc. 20:407 (1928).

2. Allen, E. J., and N. R. Angvik, "Watchdog" System Detects Chlorine Leaks at Seattle, Water Works Eng. 114:614 (1961).

3. Altman, P. L., and P. S. Dittmer (Eds.), Environmental Biology (Bethesda, Md.: Federation of Amer. Societies for Experimental Biology, 1966).

4. Arloing, E., E. Berthet, and J. Viallier, Effect of Chronic Intoxication with Chlorine at Low Concentration on Experimental Tuberculosis in Guinea Pigs, Presse Med. 48:361 (1940).

5. Atmospheric Emissions from Chlor-Alkali and Related Manufacturing Processes, U.S. Dept. of Health, Education, and Welfare, Public Health Service, National Air Pollution Control Administration and Manufacturing Chemists' Association, Inc. Report (To be published).

6. Barbour, et al., The Effects of Chlorine Upon the Body Temperature, J. Pharmacol. Expl. Therap. 14:65 (1919).

7. Barton, L. V., Toxicity of Ammonia, Chlorine, Hydrogen Cyanide, Hydrogen Sulphide, and Sulphur Dioxide Gases. I. Seeds, Contrib. Boyce Thompson Inst. 11(5):357 (1940).

8. Baskerville, C., Certain War Gases and Health, Science, 50:50 (1919).

9. Baskerville, C., Chlorine and Influenza, J. Ind. Eng. Chem. 12:293 (1920).

10. Baskerville, C., Report of the Committee on Occupational Diseases in the Chemical Trades, J. Ind. Eng. Chem. 13: 568 (1921).

11. Beck, H., Experimental Determination of Olfactory Threshold for Some Important Irritant Gases (Chlorine, Sulphur Dioxide, Ozone, Nitrosylsulphuric Acid) and Their Manifestations of the Effect of Low Concentrations on Man, Thesis, Wurzburg (1959).

12. Berghoff, R. S., The More Common Gases: Their Effect on the Respiratory Tract, Arch. Internal Med. 24:678 (1919).

13. Brennan, E., I. A. Leone, and R. H. Daines, Chlorine as a Phytotoxic Air Pollutant, Intern. J. Air Water Pollution (Oxford) 9:791 (1965).

14. Bryson, H. W., Oregon State Coll. Eng. Expt. Sta. Circ. 29:147 (1963).

15. Byers, D. H., Scientist Director, Acting Chief, Scientific Reference Service, Occupational Health Program, National Center for Urban and Industrial Health, Dept. of Health, Education, and Welfare, Public Health Service, Cincinnati, Ohio, personal communication (Dec. 1968).

16. Cady, F. H., A Kraft Mill Waste Chlorine Gas Recovery Scrubber, Presented at the Second Annual Meeting, Pacific Northwest International Section, Air Pollution Control Association, Portland, Oreg. (Nov. 5-6, 1964).

17. Chasis, H., et al., Chlorine Accident in Brooklyn, Occupational Med. 4:152 (1947).

18. Chemical Safety Data Sheet SD-80, Chlorine, Manufacturing Chemists Association, Washington, D.C. (1960).

19. Chemicals 14:2, U.S. Dept. of Commerce (June, 1967).

20. Chiarenzelli, R. V., and E. L. Joba, The Effects of Air Pollution on Electrical Contact Materials: A Field Study, J. Air Pollution Control Assoc. 16:123 (1966).

21 Chlor-Alkali Producers and Chlorine Repackagers in North America, The Chlorine Institute, Inc. (1965).

22. Chlorine, in McGraw-Hill Encyclopedia of Science and Technology (New York: McGraw-Hill, 1966).

23. Chlorine, Chemical Profile, Oil, Paint and Drug Reporter (May 1967).

24. Chlorine Manual (New York: The Chlorine Institute, Inc., 1959).

25. The Chlorine Oxidation of Sulfur Compounds in Dilute Aqueous Compounds, Atmospheric Pollution Tech. Bull. 18 (1963).

26. Cook, D. A., Test Strips for Detecting Low-Chlorine Concentrations in the Air, U.S. Patent 2,606,102 (Aug. 2, 1952).

27. Cox, H. E., Tests Available for the Identification of Small Quantities of the War Gases, Analyst 64:807 (1939).

28. Cralley, L. V., The Effect of Irritant Gases Upon the Rate of Ciliary Activity, J. Ind. Hyg. Toxicol. 24:193 (1942).

29. Current Industrial Reports, Inorganic Chemicals and Gases, U.S. Dept. of Commerce, Bureau of the Census, Washington, D.C. (1962, 1966, 1967).

30. Demidov, A. V., and L. A. Mokhov, Rapid Methods for the Determination of Harmful Gases and Vapors in the Air, Translated by B. S. Levine, U.S.S.R. Literature on Air Pollution and Related Occupational Diseases 10:114 (1962).

31. Devorkin, H., et al., Air Pollution Source Testing Manual, Air Pollution Control District, Los Angeles County, Calif. (1965).

32. Dixon, W. M., and D. Drew, Fatal Chlorine Poisoning, J. Occupational Med. 10:249 (1968).

33. Documentation of Threshold Limit Values, American Conference of Governmental Industrial Hygienists, Committee on Threshold Limit Values (1962).

34. Eichler, H., Detection of Chlorine and Bromine in Air, Gas Mixtures and Solutions by the Formation of Iris Blue, Anal. Chem. 99:272 (1934).

35. Ersepke, Z., and J. Baranek, Analyzer for Chlorine Content of Air, Chemicky Pramysl (Prague) 16(8):496 (1966).

36. Faigel, H. C., Hazards of Health Mixtures of Household Cleaning Agents, New Eng. J. Med. 271:618 (1964).

37. Fenton, P. F., Detection of War Gases, J. Chem. Educ. 21:488 (1944).

38. Ferris, B. G., Jr., W. A. Burgess, and J. Worcester, Prevalence of Chronic Respiratory Disease in a Pulp Mill and a Paper Mill in the United States, Brit. J. Ind. Med. (London) 24(1):26 (1967).

39. Fieldner, et al., Gas Masks for Gases Met in Fighting Fires, U.S. Bureau of Mines, Technical Paper 248 (1921).

40. Flake, R. E., Chlorine Inhalation, New Eng. J. Med. 271:1373 (1964).

41. Flury, F., and F. Zernik, Schadliche Gase (Berlin: Springer, 1931).

42. For Chlorine Recovery Take Your Choice, Chem. Eng. 64(6): 154 (1957).

43. Froehner, E., and R. Volker, Manual of Toxicology for Veterinaries, 6th ed. (Stuttgart: Verlag F. Enke, 1950).

44. Fukuyama, T., et al., Estimation of Toxic Gases in Air. VII. Chlorine Determination, Bull. Inst. Public Health (Tokyo) 4:10 (1955).

45. Gilchrist, H. D., and P. B. Matz, The Residual Effects of Warfare Gases; The Use of Chlorine Gas, with Reported Cases, Med. Bull. Veterans Admin. 9:229 (1933).

46. Guatelli, M. A., Analytical Procedures for the Detection of Gases, Rev. Asoc. Bioquim. Arg. 18:3 (1954).

47. Gunn, J. A., The Action of Chlorine, etc., on the Bronchi, Report to the Chemical Warfare Committee, Quart. J. Med. 13:121 (1920).

48. Haggard, H. W., Action of Irritant Gases Upon Respiratory Tract, J. Ind. Hyg. 5:390 (1923).

49. Hankin, J. L., and W. C. Klotz, Permanent Pulmonary Effects of Gas in Warfare, Am. Rev. Tuberc. 6:571 (1922).

50. Heck, W. W., et al., Environmental Pollution by Missile Propellants, Texas A. and M. Research Foundation, College Station (1962).

51. Henegar, G. P., et al., U.S. Patent 3,052,612 (1962).

52. Heyroth, F. F., "Chlorine, Cl_2," in Industrial Hygiene and Toxicology, vol. II, 2nd ed. (New York: Interscience, 1963).

53. Hindawi, I. J., Injury by Sulfur Dioxide, Hydrogen Fluoride, and Chlorine as Observed and Reflected on Vegetation in the Fields, J. Air Pollution Control Assoc. 18(5):307 (1968).

54. Hjort, A. M., and F. A. Taylor, The Effect of Morphine Upon the Alkali Reserve of the Blood of Dogs Gassed with Fatal Concentrations of Chlorine, J. Pharmacol. Exptl. Therap. 13:407 (1919).

55. Hooker, T., et al., U.S. Patent 2,750,002 (1956).

56. Hulm, R. E., U.S. Patent 2,765,873 (1956).

57. Jones, A. T., Noxious Gases and Fumes, Proc. Roy. Soc. Med. 45:609 (1952).

58. Joyner, R. E., and E. G. Durel, Accidental Liquid Spill in a Rural Community, J. Occupational Med. 4:152 (1962).

59. Karpink, R. S., U.S. Patent 2,881,054 (1959).

60. Katz, M., Sources of Pollution, Proc. Natl. Air Pollution Symp., 2nd, Pasadena, Calif. (1952).

61. Kinnear, A. M., An Alarm for the Detection of Toxic Industrial Gases, Chem. Ind. (London) 1959:361 (1959).

62. Kitagawa T., and Y. Kobayashi, Chlorine-Gas Concentration in Some Plants Manufacturing Electrolytic Sodium Hydroxide, Japan Analyst 3:42 (1954).

63. Kowitz, T. A., et al., Effects of Chlorine Gas Upon Respiratory Function, Arch. Environ. Health 14:545 (1967).

64. Kramer, C. G., Chlorine, J. Occupational Med. 9:193 (1967).

65. Krivoruchko, F. D., Determination of Chlorine in the Atmosphere, Gigiena i Sanit. 3:53 (1953).

66. Kusnetz, H. L., et al., Calibration and Evaluation of Gas Detecting Tubes, Am. Ind. Hyg. Assoc. J. 21:361 (1960).

67. Leclerc, E., and R. Haux, Modern Methods of Detection and Determination of Industrial Gases in the Atmosphere, Rev. Universelle Mines 12:293 (1936).

68. Lehmann, K. B., Experimental Studies on the Influence of Technically and Hygienically Important Gases and Vapors on the Organism, Part II and IV: Chlorine and Bromine, Arch. Hygiene 7:231 (1887).

69. Leroux, L., The Detection of Toxic Gases and Vapors, Rev. Hyg. Med. Prevent. 57:81 (1935).

70. Leukel, R. W., and O. A. Nelson, Chlorine Gas as a Seed Disinfectant, Phytopathology 29:913 (1939).

71. Liberalli, M. R., Identification of War Gases, Rev. Quim. Farm. (Rio de Janeiro) 4:49 (1939).

72. Liegel, W., and W. Oelschlager, Einfrache Methode zum Nachweis von durch Chlor an Pflanzen (Sa at, Spinat) hervorgerufenen Schadenssymptomen, Staub 22:517 (1962).

73. Litvinova, N. S., and N. Ya Khlopin, Rapid Determination of Active Chlorine in the Presence of Hydrogen Chloride in the Atmosphere of Industrial Establishments, Gigiena i Sanit. 4:45 (1953).

74. Mal'chevskii, A. N., A Method for the Rapid and Accurate Determination of Small Concentrations of Chlorine in Air, Hig. i Sanitariya 11-12:37 (1938).

75. Manufacture of Chlorine and Sodium Hydroxide, J. Air Pollution Control Assoc. 14(3):88 (1964).

76. Masterman, A. T., Color Tests for Chlorine, Ozone, and Hypochlorites with Methane Base, Analyst 64:492 (1939).

77. Matt, L., Dissertation, Wurzburg, 1881, cited in Schadliche Gase (Berlin: Springer, p. 120, 1931).

78. Maximum Permissible Concentration of Harmful Substances in Atmospheric Air Populated Places, Hyg. Sanitation 29:5 (1964).

79. McCallan, S. E. A., and C. Setterstrom, Toxicity of Ammonia, Chlorine, Hydrogen Cyanide, Hydrogen Sulphide, and Sulphur Dioxide Gases. I. General Methods and Correlations, Contrib. Boyce Thompson Inst. 11(5):325 (1940).

80. McConnaughey, P. W., Field Type Colorimetric Testers for Gases and Particulate Matter, Presented at the annual meeting of the American Industrial Hygiene Association, Atlantic City, N.J. (1958).

81. McCord, C. P., Industrial Poisoning from Low Concentrations of Chlorine Gas, J. Am. Med. Assoc. 86:1687 (1926).

82. Medical Aspects of Gas Warfare, United States Surgeon-General's Office, vol. XIV, The Medical Department of the United States Army in the World War, Prepared under the direction of Maj. Gen. M. W. Ireland, U.S. Govt. Printing Office, Washington, D.C. (1926).

83. The Merck Index, 8th ed. (Rahway, N. J.:Merck, 1968).

84. Methods for the Detection of Toxic Gases in Industry. Chlorine, Dept. of Science and Industrial Research, London, Eng. Leaflet No. 10 (1939, reprinted 1947).

85. Methods for the Detection of Toxic Substances in Air-Chlorine, Booklet No. 10, Ministry of Labor, Her Majesty's Factory Inspectorate, London (1966).

86. Miller, E. J., and F. C. Strong, A Case of Chlorine Gas Injury to Shrubs, Vines, Grass and Weeds, Arborists' News 5:73 (1940).

87. Molyneux, F., Recovery of Chlorine from By-Product Hydrochloric Acid, Chem. & Process Eng. 43:267 (1962).

88. Moureau, H., et al., Photochemical Transformation of Chloropicrin to Phosgene. I. New Reagents Sensitive and Specific for These Two Substances, Arch. Maladies Prof. Med. Travail Securite Sociale 11:445 (1950).

89. M.S.A. Chlorine-in-Air Detector, Mine Safety Appliances Co., Pittsburgh, Pa., Bull. 0805-2 (1959).

90. Patil, L. R., R. G. Smith, A. J. Vorwald, and T. F. Mooney, An Epidemiological Study of the Health of Diaphram Cell Workers Exposed to Chlorine, Presented at American Industrial Hygiene Association Meeting, Denver, Colo. (May 15, 1969).

91. Pearce, R. G., Note on Some Respiratory Studies Made on Late Stages of Gas Poisoning, Lab. Clin. Med. 5:411 (1920).

92. Pendergrass, J. A., An Air Monitoring Program in a Chlorine Plant, Am. Ind. Hyg. Assoc. J. 25(5):492 (1964).

93. Penington, A. H., War Gases and Chronic Lung Disease, Med. J. Australia 1:510 (1954).

94. Permissible Immission Concentration of Chlorine Gas, Verein Deutscher Ingineure, Kommission Reinhaltung der Luft, Duesseldorf, Germany, VDI 2106 (1960).

95. Polezhaev, N. G., Determination of Active Chlorine in the Air, U.S.S.R. Literature on Air Pollution and Related Occupational Diseases 3:29 (1960).

85. Methods for the Detection of Toxic Substances in Air-Chlorine, Booklet No. 10, Ministry of Labor, Her Majesty's Factory Inspectorate, London (1966).

86. Miller, E. J., and F. C. Strong, A Case of Chlorine Gas Injury to Shrubs, Vines, Grass and Weeds, Arborists' News 5:73 (1940).

87. Molyneux, F., Recovery of Chlorine from By-Product Hydrochloric Acid, Chem. & Process Eng. 43:267 (1962).

88. Moureau, H., et al., Photochemical Transformation of Chloropicrin to Phosgene. I. New Reagents Sensitive and Specific for These Two Substances, Arch. Maladies Prof. Med. Travail Securite Sociale 11:445 (1950).

89. M.S.A. Chlorine-in-Air Detector, Mine Safety Appliances Co., Pittsburgh, Pa., Bull. 0805-2 (1959).

90. Patil, L. R., R. G. Smith, A. J. Vorwald, and T. F. Mooney, An Epidemiological Study of the Health of Diaphram Cell Workers Exposed to Chlorine, Presented at American Industrial Hygiene Association Meeting, Denver, Colo. (May 15, 1969).

91. Pearce, R. G., Note on Some Respiratory Studies Made on Late Stages of Gas Poisoning, Lab. Clin. Med. 5:411 (1920).

92. Pendergrass, J. A., An Air Monitoring Program in a Chlorine Plant, Am. Ind. Hyg. Assoc. J. 25(5):492 (1964).

93. Penington, A. H., War Gases and Chronic Lung Disease, Med. J. Australia 1:510 (1954).

94. Permissible Immission Concentration of Chlorine Gas, Verein Deutscher Ingineure, Kommission Reinhaltung der Luft, Duesseldorf, Germany, VDI 2106 (1960).

95. Polezhaev, N. G., Determination of Active Chlorine in the Air, U.S.S.R. Literature on Air Pollution and Related Occupational Diseases 3:29 (1960).

96. Popa, I., and L. Armasescu, Determination of Gaseous Chlorine in an Industrial Atmosphere, Farmacia (Bucharest) 7:499 (1959).

97. Porter, L. E., Free Chlorine in Air. A Colorimetric Method for its Estimation, Ind. Eng. Chem. 18:730 (1926).

98. Price, G. B., After Results of Gassing, Med. Press 196: 216 (1938).

99. The Restriction of Chlorine Gas Emissions, Verein Deutscher Ingineure, Kommission Reinhaltung der Luft, Duesseldorf, Germany, VDI 2103 (1961).

100. Ronzani, E., Euber den Einfluss der Einatmungen von reinzenden Gasen der Industrien auf die Schutzhrafte des Organismus gegenuber den infektiven Krankheiten, Arch. Hyg. 70:217 (1909).

101. Rose, A., and E. Rose, The Condensed Chemical Dictionary, 7th ed. (New York: Reinhold, 1966).

102. Russkikh, A. A., Rapid Method for the Determination of Chlorine in the Atmosphere, Tr. Khim. i Khim. Tekhnol. 1(1):157 (1960).

103. Ryazanov, V. A., Limits of Allowable Concentrations of Atmospheric Pollutants, Translated by B. S. Levine, U.S. Department of Commerce, Office of Technical Services, Washington, D.C. Book 2 (1955).

104. Ryazanov, V. A., New Data on Limits of Allowable Atmospheric Pollutants, U.S.S.R. Literature on Air Pollution and Related Occupational Diseases 3:29 (1960).

105. Sandall, T. E., The Later Effects of Gas Poisoning, Lancet 2:857 (1922).

106. Schmidt, H., Beogachtung uber Gasschaden an Obstbaumen, Dt. Baumsch 3:10 (1951).

107. Schulze, E., Industrial Chlorine Gas Poisoning, J. Ind. Hyg. 21(6):139 (1939).

108. Sconce, J. S., Chlorine--Its Manufacture, Properties, and Uses, American Chemical Society Monograph Series (New York: Reinhold, 1962).

109. Silverman, L., Sampling and Analyzing Air for Contaminants, Air Conditioning, Heating Ventilating, p. 88 (1955).

110. Skljanskaja, R. M., and J. L. Rappoport, Chronic Chlorine Poisoning of Rabbits with Small Doses of Chlorine and the Development of the Fetus in Chlorine-Poisoned Rabbits, Arch. Exptl. Pathol. Pharmakol. 117:276 (1935).

111. Spector, W. S. (Ed.), Handbook of Toxicology, vol. I (Philadelphia: Saunders, 1956).

112. Stayzhkin, V. M., Hygienic Determination of Limits of Allowable Concentrations of Chlorine and Hydrochloride Gases Simultaneously Present in Atmospheric Air, Translated by B. S. Levine, U.S.S.R. Literature on Air Pollution and Related Occupational Diseases 9:55 (1962).

113. Stern, A. C. (Ed.), Air Pollution, vol. I (New York: Academic Press, p. 418, 1968).

114. Stern, A. C. (Ed.), Air Pollution, vol. II (New York: Academic Press, pp. 99,325, 1968).

115. Stern, A. C. (Ed.), Air Pollution, vol. III (New York: Academic Press, pp. 234-238, 1968).

116. Stout, G. L., Chlorine Injury to Lettuce and Other Vegetation, Calif. Dept. Agr. Month. Bull. 21:340 (1932).

117. Studinger, J., Short Scheme of Analysis for the Detection of Poison Gases, Chem. Ind. (London) 15:225 (1937)

118. Sundstrom, C., Apparatus for Detecting Chlorine Gas Leaks from Containers, U.S. Patent 2,606,101 (1952).

119. Sutter, R. C., Recovery of Chlorine from Air-Chlorine Mixtures, J. Air Pollution Control Assoc. 7:30 (1957).

120. Takhirov, M. T., Determination of Limits of Allowable Concentrations of Chlorine in Atmospheric Air, U.S.S.R. Literature on Air Pollution and Related Occupational Diseases 3:119 (1960).

121. Thomas, M. D., Gas Damage to Plants, Ann. Rev. Plant Physiol. 2:293 (1951).

122. The Thomas Register of American Manufacturers (New York: Thomas Publishing Co., 1968).

123. Thornton, N. C., and C. Setterstrom, Toxicity of Ammonia, Chlorine, Hydrogen Cyanide, Hydrogen Sulphide, and Sulphur Dioxide Gases. III. Green Plants, Contrib. Boyce Thompson Inst. 11(5):343 (1940).

124. Threshold Limit Values for 1967, Adopted at the 29th Annual Meeting of the American Conference of Governmental Industrial Hygienists, Chicago, Ill. (May 1-2, 1967).

125. Underhill, F. P. (Ed.), The Lethal War Gases; Physiology and Experimental Treatment (New Haven: Yale University Press, 1920).

126. Vedder, E. B., The Medical Aspects of Chemical Warfare (Baltimore: Williams & Wilkins, p. 70, 1925).

127. Volkening, V. B., Performance of Coating on Metal in an Industrial Atmosphere, Preprint, Presented at the 56th Annual Meeting, Air Pollution Control Association, Detroit, Mich. (June 9-13, 1963).

128. Wallach, A., and W. A. McQuary, Sampling and Determination of Chlorine in Air, Am. Ind. Hyg. Assoc. Quart. 9:63 (1948).

129. Weber, H. H., Simple Methods for the Detection and Determination of Poisonous Gases, Vapors, Smokes, and Dusts in Factory Air, Zentr. Gewerbehyg. Unvallverhuet 23:177 (1936).

130. Weedon, F. R., A. Hartzell, and C. Setterstrom, Toxicity of Ammonia, Chlorine, Hydrogen Cyanide, Hydrogen Sulphide, and Sulphur Dioxide Gases. V. Animals, Contrib. Boyce Thompson Inst. 11(5):365 (1940).

131. Wells, H., et al., Late Evaluation of Pulmonary Function After Acute Exposure to Chlorine Gas, Am. Rev. Respirat. Diseases 99:374 (1969).

132. Winternitz, M. C., Collected Studies on the Pathology of War Gas Poisoning (New Haven: Yale University Press, 1920).

133. Wright, R. H., New Work in Kraft Mill Odor Control, J. Air Pollution Control Assoc. 13(3):101 (1963).

134. Wynkoop, R., U.S. Patent 2,800,197 (1955).

135. Zais, A. M., Identification of Gas Warfare Agents, J. Chem. Educ. 21:489 (1944).

136. Zhitkova, A. S., Some Methods for the Detection and Estimation of Poisonous Gases and Vapors in the Air, A Practical Manual for the Industrial Hygienist, Service to Industry, West Hartford, Conn. (1936).

137. Zimmerman, P. W., Chemicals Involved in Air Pollution and Their Effects Upon Vegetation, Contrib. Boyce Thompson Inst. 3:124 (1955).

138. Zimmerman, P. W., Impurities in the Air and Their Influence on Plant Life, Proc. Natl. Air Pollution Symp., 1st, Pasadena, Calif., p. 135 (1949).

OTHER REFERENCES

Alekseyeva, M. V., and E. V. Elfimova, Fractional Determination of Hydrochloric Acid Aerosol and of Chlorides in Atmospheric Air, U.S.S.R. Literature on Air Pollution and Related Occupational Diseases 3:31 (1960).

American Water Works Association, Tentative AWWA Standard for Liquid Chlorine, J. Am. Water Works Assoc. 50:297 (1958).

Barnebey, H. L., Removal of Exhaust Odors from Solvent Extraction Operation by Activated Charcoal Absorption, J. Air Pollution Control Assoc. 15(9):422 (1965).

Bell, D. P., and P. C. Elmes, The Effects of Chlorine Gas on the Lungs of Rats without Spontaneous Pulmonary Disease, J. Pathol. Bacteriol. 89:307 (1965).

Benedict, H. M., and W. H. Breen, The Use of Weeds as a Means of Evaluating Vegetation Damage Caused by Air Pollution, Proc. Natl. Air Pollution Symp., 3rd, Pasadena, Calif., pp. 177-90 (1955).

Billings, C. E., C. Kurker, Jr., and L. Silverman, Simultaneous Removal of Acid Gases, Mists, and Fumes with Mineral Wool Filters, J. Air Pollution Control Assoc. 8(3):195 (1958).

Bohne, H., Air Pollution Damage from Hospital Incinerator Plants, Text in German, Staub (Dusseldorf) 27:451 (1967).

Bohne, H., Emission Damage Caused by Hospital Waste Incineration, Staub (English Translation) 27:28 (1967).

Boysen, J. E., Health Hazards of Selected Rocket Propellants, Arch. Environ. Health 7:71 (1963).

Chemical Health Hazards and Their Control: Chlorine, Quarterly Occupational Health Reports, U.S. Navy Med. News Letter 43:27 (1964).

Chlorine in Public Water Supply, Brit. Med. J. 2:1466 (1961).

Cloyd, D. R., and W. J. Murphy, Handling Hazardous Materials, National Aeronautics and Space Administration, NASA SP-5032, Sept. (1965).

Cox, R. P., Space Cabin Simulator: Atmosphere and Contaminants, Douglas Aircraft Co., Inc., Report SM-47768, Santa Monica, Calif., Feb. 25, 1965.

Dalhamn, T., Toxicity in Animal Experiments and Industrial Risks, A.M.A. Arch. Ind. Health 15:101 (1957).

Data Sheet: Chlorine, National Safety Council (1953, rev. 1966).

Eberhardt, H., European Practice in Refuse and Sewage Sludge Disposal by Incineration, Proc. Nat. Incinerator Conf. 124 (1966).

Eye Exposure to Chlorine Gas, Am. Med. Assoc. J. 165:1360 (1957).

Flour Bleachers and Improvers, Brit. Med. J. 2:631 (1961).

Gadomski, S. T., Dry-Packed Beds for the Removal of Strong-Acid Gases from Recycled Atmospheres, Naval Research Lab., Washington, D.C., Chemistry Division (1966).

Geiling, E. M. K., and F. C. McLean, Progress Report on the Toxicity of Chlorine Gas for Mice.

Goldberg, E. Kh., Photometric Determination of Small Amounts of Volatile Mineral Acids (Hydrochloric and Nitric) in the Atmosphere, Hyg. Sanitation 31(9):440 (1966).

Gross, P., W. E. Rinehart, and R. T. P. deTreville, The Pulmonary Reactions to Toxic Gases, Am. Ind. Hyg. Assoc. J. 28:315 (1967).

Gubar', M. A., and N. D. Kozlova, The Determination of Maximum Permissible Concentrations of Residual Chlorine and Chloramine in Drinking Water, Hyg. Sanitation 31:19 (1966).

Harris, L. S., Fume Scrubbing with the Ejector Venturi System, Chem. Eng. Progr. 62(4):55 (1966).

Hausberg, G., and U. Kleeberg, Installations for Purification of Waste Gases Generated During Chlorine Treatment of Light Metal Foundry Melts, Giesserei (Dusseldorf) 53(5):137 (1966).

Health Hazards of Military Chemicals, Army Chemical Warfare Laboratory, Army Chemical Center, Md. (Reports published to 1 April 1959).

Hetzel, K. W., Poisonous Action and Detection of Injurious Gases and Vapors in Mining Operations, Brennstoff-Chem. 40:115 (1959).

Hitch, J. M., Acneform Eruptions Induced by Drugs and Chemicals, Am. Med. Assoc. J. 200:879 (1967)

Jones, A. T., The Treatment of Casualties from Lung Irritant Gases with Particular Reference to the Use of Oxygen and Carbon Dioxide Mixture, J. Ind. Hyg. & Toxicol. 22:235 (1940).

Knott, K. H., and S. Turkolmez, Krupp Rotary Brush Scrubber for the Control of Gas, Vapour, Mist and Dust Emissions, Krupp Tech. Rev. (Essen) 24 (1):25 (1966).

Konig, W., Chemical Warfare Agents (Berlin: German Military Pub. House, 1964).

Koski, T. A., L. S. Stuart, and L. F. Ortenzio, Comparison of Chlorine, Bromine, and Iodine as Disinfectants for Swimming Pool Water, Appl. Microbiol. 14:276 (1966).

Krivoruchko, F. D., Determination of Aerial Concentration of Decomposition and Oxidation Products of Certain Organoaluminum Compounds (TEA, DEAC, TIBA, and DIBAC), Hyg. Sanitation 31(8): 256 (1966).

Laubusch, E. J., Water Disinfection Practices in the United States, J. Am. Water Works Assoc. 52:1416 (1960).

Lodge, J. P., Jr. (Ed.) Atmospheric Chemistry of Chlorine and Sulfur Compounds; Proceedings of a Symposium, Cincinnati, Ohio, 1957, Geophysical Monograph No. 3, Natl. Acad. Sci.-- Natl. Res. Council Publ. 652.

Nehrkorn, A. and W. Schwartz, On Disappearance of Chlorine in Drinking Water. I. Behavior of Plastic Piping with Reference to Chlorine Dimunition, Arch. Hyg. Bakteriol. 145:481 (1961).

Noe, J. T., Therapy for Chlorine Gas Inhalation, Ind. Med. Surg. 32:411 (1963).

Petrie, E. M., and D. P. Roman, Chlorine Sanitizing Compounds, Soap and Chem. Specialties 34:67 (1958).

Report of Working Group Meeting. Encl: Limits for Atmospheric Constituents in Nuclear Submarines, Working Group for Submarine Atmosphere Requirements, Naval Ship Engineering Center (Mar. 8, 1967).

Rose, G., Will Trash Removal be a Marketing Factor for the Glass Container Industry and Producers of Other Packaging Material?, Glastech. Ber. (Frankfurt) 40(11):438 (1967).

Sayers, R. R., Toxicology of Gases and Vapors, Intern. Crit. Tables 2:318 (1927).

Sherwood, P. W., How Atmospheric Conditions Can Corrode Refinery Equipment, Erdoel Kohle (Hamburg) 19(4):289 (1966).

Smyth, H. F., Jr., Sufficient Challenge, Fd. Cosmet. Toxicol. 5:51 (1967).

Thomas, M. D., Effects of Air Pollution on Plants, World Health Organization Monograph Series, 46 (Air Pollution), p. 233 (1961).

Tubb, C. M., Chlorine--The Ubiquitous Halogen, Chem. Ind. 1963:1421 (1963).

Uchigasaki, K., et. al., Absorption of Gases in Liquid with Corona Discharge, Text in Japanese, Chem. Eng. (Tokyo) 31:878 (1967).

Viola, P. L., Influence of Chlorine on the Poisoning by Mercury Vapour, Med. Lavoro 58:60 (1960).

Water Chlorination and Hepatitis, Am. Med. Assoc. J. 167:2153 (1958).

Yanysheva, N., The Effect of Atmospheric Air Pollution by Discharges from Electric Power Plants and Chemical Combines on the Health of Nearby Inhabitants, Translated by B. S. Levine, U.S.S.R. Literature on Air Pollution and Related Occupational Diseases 1:98 (1960).

APPENDIX

APPENDIX

TABLE 1

REACTIVITY OF CHLORINE[18,24]

Flammability:	Chlorine is nonflammable; however, like oxygen gas, it is capable of supporting combustion of many materials including hydrogen, reactive metals, and many organic compounds.
Reaction with water:	Chlorine reacts with water to produce hydrochloric acid and hypochlorous acid (see Equation 1); the latter will decompose (slowly at normal temperatures) to form hydrochloric acid and oxygen (see Equation 2). A basic solution will tend to drive the first reaction to the right (Equation 1), whereas acid tends to drive the reaction to the left or produce free chlorine gas. The second reaction is irreversible. Chlorine may also form a hydrate ($Cl_2 \cdot 8H_2O$) below 49.3°F.
	(Equation 1) $Cl_2 + H_2O \rightleftharpoons HCl + HClO$
	(Equation 2) $2HClO \rightarrow 2HCl + O_2$
Reaction with metals:	Dry chlorine reacts with many metals--including aluminum, gold, mercury, selenium, and tin--but is unreactive to copper, iron, lead, nickel, and steel at normal temperatures.
	Moist chlorine reacts with almost all metals except gold, platinum, and titanium.
Reaction with non-metallic elements:	Chlorine reacts with most other elements, sometimes very rapidly. It is unreactive to oxygen and nitrogen at normal temperatures.
Reaction with compounds:	Chlorine reacts with basic materials to form chlorides and hypochlorites (e.g., see Equation 3 for reaction with sodium hydroxide). Chlorine has a great affinity for a compound containing hydrogen to yield hydrogen chloride. Many organic compounds are extremely reactive, yielding organic chlorinated derivatives and hydrochloric acid.
	(Equation 3) $Cl_2 + 2NaOH \rightleftharpoons NaCl + NaOCl + H_2O$

APPENDIX

TABLE 2

PHYSICAL PROPERTIES OF CHLORINE[18,83]

Molecular formula:	Cl_2 (diatomic molecule)
Atomic number:	17
Molecular weight:	70.914
Atomic weight:	35.457
Isotopes (abundance):	35 (75.53%) 37 (24.47%)
Boiling point:	-34.05°C (-29.29°F)
Melting point:	-100.98°C (-149.76°F)
Color, gas:	Greenish yellow
Color, liquid:	Clear amber
Odor:	Characteristic: penetrating and irritating
Density, gas, dry, 0°C:	3.209 g/liter (0.2003 lb/ft^3)
Density, gas, saturated, 0°C:	12.07 g/liter (0.7537 lb/ft^3)
Density, liquid (-33.6°C):	1.557 g/liter
Density, liquid (0°C):	1,468 g/liter (91.67 lb/ft^3)
Solubility, 10°C:	1 vol H_2O dissolves 2.7 vol Cl_2 (0.8% by wt)
Solubility, 20°C:	1 vol H_2O dissolves 2.3 vol Cl_2 (0.7% by wt)
Solubility, 30°C:	1 vol H_2O dissolves 1.8 vol Cl_2 (0.5% by wt)
Vapor pressure, -10°F:	8.29 psig*
Vapor pressure, 0°F	13.81 psig*
Vapor pressure, 20°F:	27.84 psig*
Vapor pressure, 60°F:	70.91 psig*
Vapor pressure, 80°F:	101.76 psig*

*psig: pounds per square inch gauge.

APPENDIX

TABLE 3

SUMMARY OF REPORTED HUMAN HEALTH EFFECTS OF INHALATION OF CHLORINE

Concentration (ppm)	Exposure Time	Effects or Comments	Reference
<1.0		Disturbances and objective symptoms of irritation created at this concentration	94
1		None	85
1-2		Work without interruption possible	52, 77
1		Least amount required to produce slight symptoms after several hours' exposure	39
3.3	Several hours	Risk to health or life; impossible working conditions	94
3-5		Tolerable for short periods of time without objective evidence of injury	64
3-6		Stinging or burning sensation present in the eyes, nose, and throat; sometimes headache due to irritation of the accessory nasal sinuses	41, 52
4		Maximum amount that can be inhaled for 1 hour without serious disturbances	39
4		Slight smarting of the eyes and irritation of the nose and throat	85

(continued

APPENDIX

TABLE 3 (Continued)

SUMMARY OF REPORTED HUMAN HEALTH EFFECTS OF INHALATION OF CHLORINE

Concentration (ppm)	Exposure Time	Effects or Comments	Reference
~5	Working conditions	Premature aging; those exposed suffer from disease of bronchi and become predisposed to tuberculosis; teeth corrode from hydrochloric acid produced in mouth; inflammation or ulceration of the mucous membrane of nose.	52, 100
5	30-60 min	Does not endanger life	94
5		Noxious effect; impossible to breathe several minutes	39
10	<1 min	Severe coughing and eye irritation	85
>10		Immediate and delayed effects; may be serious	85
14-21	0.5-1.0 hr	Dangerous	52, 126
15.1		Least amount required to cause irritation of throat	39
20	<30 min	Endangers life	94
30.2		Least amount required to cause coughing	39
40-60		Amount dangerous in 30 minutes to 1 hour	18
50	30-60 min	Fatal immediately or eventually	94
100	<1 min	Cannot be tolerated for longer than 1 minute	52

APPENDIX

TABLE 4

SUMMARY OF REPORTED TOXIC EFFECTS OF INHALATION OF CHLORINE ON ANIMALS

Species	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
Animals	~1.5		Generally still tolerable for animals	40, 94
Animals	20.7		No damage when repeatedly exposed	52, 94
Animals	~60		Exposure causes sickness	43, 94
Animals	200-1,000		Respiratory rate increases during exposure	47, 52
Animals	~600		Death occurs	43, 94
Animals	1,000		Brief exposure kills even large animals	41, 52
Cat	10-100		Threshold values injurious to health depending on duration of exposure	68, 94
Cat	280-630	Continuous	Death after 60 minutes of exposure	68, 111
Cat	300	60 min	May cause death after a period during which conjunctiva is inflamed and there is coughing and dyspnea	52

(continued)

APPENDIX

TABLE 4 (Continued)

SUMMARY OF REPORTED TOXIC EFFECTS OF INHALATION OF CHLORINE ON ANIMALS

Species	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
Dog	50-2,000	30 min	Decrease, followed by increase, of pulse rate; increase in respiratory frequency; initial decrease in temperature; respiratory acidosis	3, 125
Dog	180-200 (or more)		Pulse rate is retarded during exposure	6, 52
Dog	<280	30 min	Death never occurs	52
Dog	<650	30 min	Death rarely occurs	52
Dog	800	2.7 hr	Rapidly increasing acidosis	52, 54
Dog	800-900	30 min	Death occurred sometime after exposure. Rapid and acute death of 50% of dogs. Autopsy indicated edema in the lungs and necrosis of the bronchial epithelium	6, 82 111, 130
Guinea pigs			Small quantities accelerate the course of experimental tuberculosis	4, 52
Guinea pigs	280-630	Continuous	Death after 64 minutes of exposure	68, 111

(continued)

APPENDIX

TABLE 4 (Continued)

SUMMARY OF REPORTED TOXIC EFFECTS OF INHALATION OF CHLORINE ON ANIMALS

Species	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
House flies	16	>960 min	Approximately 50% died	130
House flies	63	840 min	Approximately 50% died	130
House flies	250	240 min	Approximately 50% died	130
House flies	1,000	45 min	Approximately 50% died	130
House flies	1,000	10 min	Approximately 35% died	130
House flies	1,000	1 hr	Approximately 76% to 91% died	130
Mice	63	>960 min	Approximately 50% died	130
Mice	250	440 min	Approximately 50% died	130

(continued)

APPENDIX

TABLE 4 (Continued)

SUMMARY OF REPORTED TOXIC EFFECTS OF INHALATION OF CHLORINE ON ANIMALS

Species	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
Mice*	1,000	28 min	Approximately 50% died	130
Rabbit (Excised trachea)	20-200	0.5 to 2 min	Arrest of ciliary activity	130
Rabbits	~0.7 - 1.7	Up to 9 mos	Caused loss of weight and an increased incidence of respiratory disease. Catarrh-like changes observed in upper respiratory tract, as well as lung hemorrhages and emphysema.	3, 28
Rats*	63	>960 min	Approximately 50% died	130
Rats*	250	440 min.	Approximately 50% died	130
Rats*	1,000	53 min	Approximately 50% died	130

*Autopsy data on exposed rats and mice are given in Table 5.

APPENDIX

Table 5

TYPICAL GROSS FINDINGS AT AUTOPSY OF RATS AND MICE WHICH DIED DURING EXPOSURE TO CHLORINE (Cl_2) OR WERE SACRIFICED IMMEDIATELY AFTER GAS TREATMENT[130]

Organs	Concentration of Chlorine Gas				
	3,000,000 µg/m³ (1,000 ppm)		750,000 µg/m³ (250 ppm)		189,000 µg/m³ (63 ppm)
	Rats	Mice	Rats	Mice	Rats*
Brain	Slightly congested	Not congested	Slightly congested	Slightly congested	Slightly congested
Trachea	Not reddened	Not reddened	Not reddened	Not reddened	Not reddened
Lungs	Distended, filling cavity; pale, waxy cut surfaces; foamy	Partly collapsed and hemorrhagic	Distended; pale and waxy, with scattered hemorrhages; wet	Deep black-red, cut surfaces dripping blood	Distended; pink, very rare punctate hemmorrhages; cut surfaces wet and foamy
Heart	Greatly distended on right side, atria distended	In systole or moderately distended	Right side slightly dilated	In systole	Right side distended
Liver	Congested	Congested	Moderately congested	Twice natural size; waxy pale, nutmeg color	Pale to dark red in color
Gall bladder	Not distended	Not distended	Not distended	Not distended	Not distended
Stomach	Moderately to greatly distended, few small hemorrhages	Same as rats	Greatly distended, very rare small hemorrhages	Moderately distended	Distended, very rare punctate hemorrhages
Intestines	Large and small, partly distended	Natural	Large and small, moderately distended	Slightly distended	Small intestine, moderately distended; colon moderately distended.
Adrenals	Pink	Pink	Pale	Pale	Pale to natural pink in color
Kidneys	Congested	Congested	Congested	Pale	Pale to dark red in color

*Autopsied immediately after exposure.

APPENDIX

TABLE 6

SUMMARY OF REPORTED EFFECTS OF CHLORINE GAS EXPOSURE ON PLANT LIFE

Plant	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
Alfalfa	0.1	2 hr	Incipient injury	13
Beans	1.3	30 min	Incipient injury	121, 138
Buckwheat	0.46	60 min	Incipient injury	121, 138
Buckwheat leaves	1,000	<4 min	50% injury to exposed area	123
Buckwheat stems	1,000	120 min	50% injury to exposed area	123
Azalea	0.8	4 hr	Incipient injury	13
Begonia	1.0	4 hr	No visible injury	13
Chenopodium	1.0	4 hr	No visible injury	13
Chinese holly (*Ilex cornuta Lindl*)	0.46 - 4.67	3 hr	No visible damage	138

(continued)

APPENDIX

TABLE 6 (Continued)

SUMMARY OF REPORTED EFFECTS OF CHLORINE GAS EXPOSURE ON PLANT LIFE

Plant	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
Coleus (*Coleus blumei* Benth)	0.56	120 min	Incipient injury	138
Corn	0.10 - 0.25	4 hr	Incipient injury	13
Corn	62	25 min	Incipient injury: interveinal browning of older leaves	50
Corn	62	60 min	Severe damage: all plants (two) died	50
Cucumber	0.50	4 hr	Incipient injury	13
Corn	62	180 min	Severe damage: all plants (six) died	50
Cowpea	0.80	4 hr	Incipient injury	13
Cowpea	62	13 min	Incipient injury: loss of turgor and interveinal necrosis	50

(continued)

APPENDIX

TABLE 6 (Continued)

SUMMARY OF REPORTED EFFECTS OF CHLORINE GAS EXPOSURE ON PLANT LIFE

Plant	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
Cowpea	62	60 min	Moderate damage but plants survived	50
Cowpea	62	180 min	Severe damage but plants survived	50
Cotton	62	13 min	Incipient injury: brown necrosis of cotyledons and leaves along margins	50
Cotton	62	60 min	Moderate damage but plants survived	50
Cotton	62	180 min	Severe damage but plants survived	50
Dahlia	0.50	4 hr	Incipient injury	13
Dandelion	0.50	4 hr	Incipient injury	13
Eggplant	0.46 - 4.67	180 min	No visible damage	138

(continued)

APPENDIX

TABLE 6 (Continued)

SUMMARY OF REPORTED EFFECTS OF CHLORINE GAS EXPOSURE ON PLANT LIFE

Plant	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
Geranium	0.80	4 hr	Incipient injury	13
Gomphrena	0.10-0.25	4 hr	Incipient injury	13
Halsea sp.	0.56	120 min	Incipient injury	138
Mustard	0.10 - 0.25	4 hr	Incipient injury	13
Nasturtium	0.50	4 hr	Incipient injury	13
Oxalis	1.0	4 hr	No visible damage	13
Onion	0.10 - 0.25	4 hr	Incipient injury	13
Peach	0.56	180 min	Incipient injury	121, 138
Pepper	1.0	4 hr	No visible injury	13
Petunia	0.80	4 hr	Incipient injury	13

(continued)

APPENDIX

TABLE 6 (Continued)

SUMMARY OF REPORTED EFFECTS OF CHLORINE GAS EXPOSURE ON PLANT LIFE

Plant	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
Polygonum	1.0	4 hr	No visible injury	13
Pinto bean	0.80	4 hr	Incipient injury	13
Radish (*Raphanus sativus* L.)	1.3	30 min	Incipient injury	121, 138
Radish	0.1	2 hr	Incipient injury	13
Radish seeds, dry	250 and 1,000	1 - 960 min	Germination not affected	7
Radish seeds, soaked	250 - 1,000	1 - 240 min	Germination not affected	7
	250	960 min	Germination reduced by about 15-20%	
	1,000	960 min	Germination reduced by about 90-95%	
Rhodotypos sp.	1.3	30 min	Incipient injury	138

(continued)

APPENDIX

TABLE 6 (Continued)

SUMMARY OF REPORTED EFFECTS OF CHLORINE GAS EXPOSURE ON PLANT LIFE

Plant	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
Roses (Rosa)	1.5	30 min	Incipient injury	121, 138
Rye seeds, dry	250-1,000	1-240 min	Little effect on germination	7
	250	960 min	Germination reduced by approximately 10%	
	1,000	960 min	Germination reduced by approximately 50%	
Rye seeds, soaked	250	1-60 min	Little effect on germination	7
	1,000	1 min	" " "	
	250	240 min	Germination reduced by approximately 26%	
	250	960 min	Germination reduced by approximately 68% and bleaching of seeds	
	1,000	60 min	Germination reduced by approximately 20%	
	1,000	240 min	" " 70%	
	1,000	960 min	No germination and bleaching of seeds	

(continued)

TABLE 6 (Continued)

SUMMARY OF REPORTED EFFECTS OF CHLORINE GAS EXPOSURE ON PLANT LIFE

Plant	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
Scotia bean	0.80	4 hr	Incipient injury	13
Squash	0.80	4 hr	Incipient injury	13
Squash	62	13 min	Incipient injury: loss of turgor, interveinal necrosis of young mature leaves	50
	62	60 min	Severe damage but plants survived	
	62	180 min	All plants died (total of six)	
Soybean	62	13 min	Incipient injury: loss of turgor	50
	62	60 min	Moderate injury but plants survived	
	62	180 min	Severe injury but plants survived	
Sunflower	0.10-0.25	4 hr	Incipient injury	13

(continued)

APPENDIX

TABLE 6 (Continued)

SUMMARY OF REPORTED EFFECTS OF CHLORINE GAS EXPOSURE ON PLANT LIFE

Plant	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
Tobacco	0.10-0.25	4 hr	Incipient injury	13
Tobacco leaves	1,000	0.5 min	50% injury to exposed areas	123
Tobacco stems	1,000	60 min	50% injury to exposed areas	123
Tomato	0.5	4 hr	Incipient injury	13
Tomato leaves	0.40	6 hr	No visible injury to leaves	13
	0.60	2 hr	No visible injury to leaves	
	0.6	3 hr	Moderate injury to leaves	
Tomato leaves	1.4	2 hr	Severe injury to leaves	13, 123
	1.4	1 hr	Moderate injury to leaves	
	1,000	0.8 min	50% injury to exposed area	
	1,000	~0.6 min*	Clear weather: 50% injury to exposed area	
	250	~0.6 min*	" " "	

(continued)

APPENDIX

TABLE 6 (Continued)

SUMMARY OF REPORTED EFFECTS OF CHLORINE GAS EXPOSURE ON PLANT LIFE

Plant	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
Tomato leaves	63	~0.8 min*	Clear weather: 50% injury to exposed area	13, 123
	4	~9.5 min*	" " "	
	1,000	~1 min*	Cloudy weather: 50% injury to exposed area	
	250	~4 min*	" " "	
	16	~120 min*	" " "	
Tomato stems	1,000	22 min	50% injury to exposed area	123
	1,000	~150 min*	Clear weather: 50% injury to exposed area	
	250	~35 min*	" " "	
	63	~520 min*	" " "	
	4	~240-600 min*	" " "	
	1,000	~9.5 min*	Cloudy weather: 50% injury to exposed area	
	250	>960 min*	" " "	
	16	~520 min*	" " "	

(continued)

TABLE 6 (Continued)

SUMMARY OF REPORTED EFFECTS OF CHLORINE GAS EXPOSURE ON PLANT LIFE

Plant	Concentration (ppm)	Exposure Time	Effects or Comments	Reference
Wheat, oat, and barley seeds	3,000-9,000	1-2 hr	No visible injury	70
Zinnia	0.10-0.25	4 hr	Incipient injury	13

*Data compiled from a graph.

APPENDIX

TABLE 7

CHLORINE PRODUCTION IN THE UNITED STATES[29]
(Short Tons per Year)

Year	Liquid	Gas*
1958	2,000,318	3,604,538
1959	2,233,329	4,347,118
1960	2,398,031	4,636,939
1961	2,478,007	4,600,791
1962	2,755,162	5,142,876
1963	2,920,127	5,464,080
1964	3,120,201	5,945,215
1965	3,484,312	6,517,079
1966	3,853,628	7,205,165
1967	3,950,540	7,653,881

*The figure given for gas includes the figure given for liquid.

APPENDIX

TABLE 8

MAJOR PRODUCERS OF CHLORINE GAS AND LIQUID IN THE U.S.[122]

Company	Location
Adirondack Chemical Co.	Plattsburgh, N.Y.
Allied Chemical Corp., Industrial Chemicals Div.	Morristown, N.J.
American Oil & Supply Co.	Newark, N.J.
Brown Co.	New York, N.Y.
Calvert Chemical Co.	Cincinnati, Ohio
Diamond Alkali Co.	Cleveland, Ohio
Dow Chemical Co.	Midland, Mich.
Field Point Mfg. Corp.	Providence, R.I.
FMC Corp., Inorganic Chemicals Div.	New York, N.Y.
General Aniline & Film Corp.	New York, N.Y.
General Biochemicals, Inc.	Chagrin Falls, Ohio
Georgia-Pacific Corp.	Portland, Oreg.
Globe Chemical Co., Inc.	Cincinnati, Ohio
Haviland Products Co.	Grand Rapids, Mich.
Hooker Chemical Corp., Industrial Chemicals Div.	Niagara Falls, N.Y.
Hubbard-Hall Chemical Co.	Waterbury, Conn.
Jones Chemicals Inc.	Caledonia, N.Y.
Joseph, E. R., Co., Inc.	Norristown, Pa.
Kingston Chemical Co., Inc.	New York, N.Y.
Modern Pool Products, Inc.	Stamford, Conn.
Ocean Pool Supply Co.	Huntington Station, Long Island, N.Y.
Olin Mathieson Chemical Corp., Chemicals Div.	New York, N.Y.
Peebles Chemical Co.	Kenilworth, Md.
Pennsalt Chemical Corp.	Philadelphia, Pa.
Phillip Brothers Chemicals, Inc.	New York, N.Y.
PPG Industries, Chemical Div.	Pittsburgh, Pa.
Precision Gas Products, Inc.	Linden, N.J.
Robinson Bros. Chemicals, Inc.	Brooklyn, N.Y.
Seaway Chemical Corp.	Buffalo, N.Y.
Sergeant, E. M., Pulp & Chemical Co., Inc.	New York, N.Y.
Sherwood Overseas Corp.	New York, N.Y.
Stauffer Chemical Co., Industrial Chemical Div.	New York, N.Y.
Tesco Chemicals, Inc.	Atlanta, Ga.
Vulcan Materials Co.	Birmingham, Ala.
Wittichen Chemical Co.	Birmingham, Ala.
Wyandotte Chemicals Corp., Industrial Chemicals Group	Wyandotte, Mich.

TABLE 9

CONSUMPTION OF CHLORINE BY USES, 1963-64[19]

Use	Percent of Total 1963	1964
1. Vinyl and vinylidine chloride monomers	7.6[a]	8.8[a]
2. Other chlorinated hydrocarbons	31.8	31.4
3. Glycerine and glycols	14.0	14.6
4. Metal alkyds, including tetra-ethyl lead or tetramethyl lead	[b]	[b]
5. Bleaching compounds (excludes quantities used for pulp and paper and textile bleaching and finishing)	2.0 2.0	2.1
6. Phosphates	1.0	0.5
7. Silicates	0	0
8. Soap and synthetic detergents, other than above	0.2	[c]
9. Pesticides	4.2	4.0
10. Other chemicals	10.9[d]	10.8[d]
11. Rubber reclaiming processes	0.1	0.2
12. Water and sewage treatment	1.2[e]	1.2[e]
13. Pulp and paper manufacture	15.4	14.9
14. Textile bleaching and finishing	0.1	0.1
15. Glass	0	0
16. Cellophane	[c]	[c]
17. Rayon	[c]	[c]
18. Aluminum and other production and ore-treating	0.3	0.3
19. Food	0.4	0.4
20. Pharmaceuticals	0.1	0.1
21. Petroleum production and refining	0.2	0.2
22. Other uses	6.1[d,f]	6.1[d,f]
23. Sales to dealers	3.6	3.4
24. Sales to government	0.3	0.3
25. Exports	0.5	0.6
Total consumption (in thousands of short tons)	5,576	6,096

[a]Probably greater; some chlorine intended for plastics and chlorinated organics was reported under Item 10.

[b]Withheld to avoid disclosure of figures for individual companies.

[c]Less than 0.05 percent

[d]Items 10 and 22 also include such uses as for aluminum, ammonium and calcium chlorides, boric acid, bromine, rubber chemicals, paint coatings, ferric chloride, etc.

[e]Probably greater, approximately 3.5 percent; some chlorine for water and waste treatment was included in Items 10, 22, 23.